ESC European Society of Cardiology

European Heart Journal (2025) **00**, 1–107
https://doi.org/10.1093/eurheartj/ehaf193

ESC GUIDELINES

2025 ESC Guidelines for the management of cardiovascular disease and pregnancy

Developed by the task force on the management of cardiovascular disease and pregnancy of the European Society of Cardiology (ESC)

Endorsed by the European Society of Gynecology (ESG)

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Councils: Council on Cardiovascular Genomics, Council on Hypertension.

Working Groups: Adult Congenital Heart Disease, Aorta and Peripheral Vascular Diseases, Cardiovascular Pharmacotherapy, Cardiovascular Surgery, Myocardial and Pericardial Diseases, Pulmonary Circulation and Right Ventricular Function, Thrombosis.

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SD All experts involved in the development of these guidelines have submitted declarations of interest which are reported in a supplementary document to the guidelines. See the *European Heart Journal* online or https://www.escardio.org/Guidelines for supplementary documents as well as evidence tables.

Keywords Guidelines • Pregnancy • Cardiovascular • Heart failure • Arrhythmias • Genetic counselling • Risk stratification • Pregnancy heart team • Cardiomyopathies • Drugs in pregnancy • Aortic disease • Adult congenital heart disease • Peripartum cardiomyopathy • Hypertensive disorders of pregnancy • Adverse pregnancy outcomes

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Abbreviations and acronyms

ACE-(I)	Angiotensin-converting enzyme(-inhibitor)
ACHD	Adult congenital heart disease
ACS	Acute coronary syndrome
ADR	Adverse drug reaction
AF	Atrial fibrillation
AFL	Atrial flutter
AHF	Acute heart failure
AHT	Arterial hypertension
ALARA	As low as reasonably achievable
ALT	Alanine transaminase
AMVP	Arrhythmic mitral valve prolapse
APO	Adverse pregnancy outcomes
aPTT	Activated partial thromboplastin time
ARB	Angiotensin receptor blocker
ARNI	Angiotensin receptor/neprilysin inhibitor
ARVC	Arrhythmogenic right ventricular cardiomyopathy
ASA	Acetylsalicylic acid
ASCVD	Atherosclerotic cardiovascular disease
ASD	Atrial septal defect
ASI	Aortic size index
AST	Aspartate aminotransferase
AV	Atrioventricular
AV(N)RT	Atrioventricular (nodal) re-entry tachycardia
AVSD	Atrioventricular septal defect
BAV	Bicuspid aortic valve
B-blocker	Beta-blocker
b.i.d.	Bis in die (twice a day)
BMI	Body mass index
BNP	B-type natriuretic peptide
BP	Blood pressure
b.p.m.	Beats per minute
BrS	Brugada syndrome
CABG	Coronary artery bypass graft
CAD	Coronary artery disease
CARPREG II	Cardiac Disease in Pregnancy study II
CCB	Calcium channel blocker
CCS	Chronic coronary syndromes
CI	Confidence interval
CMP	Cardiomyopathy
CMR	Cardiovascular magnetic resonance

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CO	Cardiac output
COR	Class of recommendation
CPG	Clinical Practice Guidelines
CPR	Cardiopulmonary resuscitation
CPVT	Catecholaminergic polymorphic ventricular tachycardia
CT	Computed tomography
cTnI	Cardiac troponin I
cTnT	Cardiac troponin T
CTPA	Computed tomography pulmonary angiography
CTRCD	Cancer-therapy-related cardiac dysfunction
CVD	Cardiovascular disease
DAPT	Dual antiplatelet therapy
DCM	Dilated cardiomyopathy
DOAC	Direct oral anticoagulant
DVT	Deep vein thrombosis
ECG	Electrocardiogram
ECMO	Extracorporeal membrane oxygenation
EF	Ejection fraction
EORP	EURObservational Research Programme
ERA	Endothelin receptor antagonist
ESC	European Society of Cardiology
ESH	European Society of Hypertension
FAT	Focal atrial tachycardia
FDA	Food and Drug Administration
FFP	Fresh frozen plasma
GDM	Gestational diabetes mellitus
HCM	Hypertrophic cardiomyopathy
HELLP	Haemolysis, elevated liver enzymes, low platelet count
HF	Heart failure
HFrEF	Heart failure with reduced ejection fraction
HLA	Human leucocyte antigen
HR	Heart rate
HTAD	Heritable thoracic aortic disease
ICD	Implantable cardioverter defibrillator
INR	International normalized ratio
IU	International unit
IUGR	Intrauterine growth restriction
IUD	Intrauterine device
i.v.	Intravenous
IVF	*In vitro* fertilization
LA	Left artery
LDL	Low-density lipoprotein
LDS	Loeys–Dietz syndrome
LMWH	Low-molecular-weight heparin
LQT1/2/3	Long QT syndrome type 1, 2, or 3
LQTS	Long QT syndrome
LV	Left ventricle
LVAD	Left ventricular assist device
LVEDD	Left ventricular end-diastolic diameter
LVEF	Left ventricular ejection fraction
LVH	Left ventricular hypertrophy
LVOT(O)	Left ventricular outflow tract (obstruction)
MACE	Major adverse cardiovascular events
MFS	Marfan syndrome
MHV	Mechanical heart valve
MINOCA	Myocardial infarction with non-obstructive coronary arteries
MR	Mitral valve regurgitation
MRA	Mineralocorticoid receptor antagonist
mU	Milliunit
mWHO	modified World Health Organization
NA	Not applicable
NDLVC	Non-dilated left ventricular cardiomyopathy
NP	Natriuretic peptide (NT-proBNP and BNP)
NSAID	Non-steroidal anti-inflammatory drug
nsHTAD	Non-syndromic heritable thoracic aortic disease
NSTE ACS	Non-ST-elevation acute coronary syndrome
NT-proBNP	N-terminal pro-brain natriuretic peptide
NYHA	New York Heart Association
o.d.	Omni die (once a day)
oGTT	Oral glucose tolerance test
OR	Odds ratio
P/LP	Pathogenic/likely pathogenic
PAH	Pulmonary arterial hypertension
PAP	Pulmonary arterial pressure
PASP	Pulmonary arterial systolic pressure
PCI	Percutaneous coronary intervention
PE	Pulmonary embolism
PH	Pulmonary hypertension
PlGF	Placental growth factor
p.o.	Per os (by mouth)
PPCM	Peripartum cardiomyopathy
PPH	Post-partum haemorrhage
PREM	Patient-reported experience measure
PROM	Patient-reported outcome measure
PV	Pulmonary valve
QRS	Q, R, and S waves
RBC	Red blood cell
RID	Relative infant dose
ROPAC	Registry of Pregnancy and Cardiac Disease
ROSC	Return of spontaneous circulation
RV	Right ventricle
RVEF	Right ventricular ejection fraction
RVH	Right ventricular hypertrophy
RVOT(O)	Right ventricular outflow tract (obstruction)
RVOT-VT	Right ventricular outflow tract ventricular tachycardia
SBP	Systolic blood pressure
SCAD	Spontaneous coronary artery dissection
SGA	Small for gestational age
SGLT2	Sodium–glucose co-transporter-2
SPAP	Systolic pulmonary artery pressure
SpO_2	Oxygen saturation
SQTS	Short QT syndrome
STEMI	ST-elevation myocardial infarction
SVT	Supraventricular tachycardia
TAD	Thoracic aortic disease
TAVI	Transcatheter aortic valve implantation
TGA	Transposition of the great arteries
TOF	Tetralogy of Fallot
TPVI	Transcatheter pulmonary valve implementation
TR	Tricuspid regurgitation
TS	Turner syndrome
TTE	Transthoracic echocardiogram
UACR	Urine albumin–creatinine ratio
UFH	Unfractionated heparin

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VA-ECMO	Veno-arterial extracorporeal membrane oxygenation
VF	Ventricular fibrillation
VHD	Valvular heart disease
VKA	Vitamin K antagonist
VSD	Ventricle septal defect
VT	Ventricular tachycardia
VTE	Venous thromboembolism
WCD	Wearable cardioverter defibrillator
WHO	World Health Organization
WPW	Wolff–Parkinson–White

1. Preamble

Guidelines evaluate and summarize available evidence with the aim of assisting health professionals in proposing the best diagnostic or therapeutic approach for an individual patient with a given condition. European Society of Cardiology (ESC) Guidelines are intended for use by health professionals but do not override their individual responsibility to make appropriate and accurate decisions in consideration of each patient's health condition and in consultation with the patient or the patient's caregiver where appropriate and/or necessary. It is also the health professional's responsibility to verify the rules and regulations applicable in each country to drugs and devices at the time of prescription and to respect the ethical rules of their profession.

ESC Guidelines represent the official position of the ESC on a given topic. Guideline topics are selected for updating after annual expert review of new evidence conducted by the ESC Clinical Practice Guidelines (CPG) Committee. European Society of Cardiology Policies and Procedures for formulating and issuing ESC Guidelines can be found on the ESC website (https://www.escardio.org/Guidelines/Clinical-Practice-Guidelines/Guidelines-development/Writing-ESC-Guidelines).

This guideline updates and replaces the previous version from 2018. This Task Force was selected by the ESC to include professionals involved with the medical care of patients with this pathology and to include patient representatives and methodologists. The selection procedure included an open call for authors and aimed to include members from across the whole of the ESC region and from relevant ESC Subspecialty Communities. Consideration was given to diversity and inclusion.

Guidelines Task Forces perform a critical review and evaluation of the published literature on diagnostic and therapeutic approaches including assessment of the risk–benefit ratio. Recommendations are based on major randomized trials and relevant systematic reviews and meta-analyses, when available. Systematic literature searches are conducted in cases of controversy or uncertainty to ensure that all key studies were considered. For recommendations related to diagnosis and prognosis, additional types of evidence are included, such as diagnostic accuracy studies and studies focused on the development and validation of prognostic models. The strength of each recommendation and the level of evidence supporting it are weighed and scored according to predefined criteria as outlined in *Tables 1 and 2*. Patient-reported outcome measures (PROMs) and patient-reported experience measures (PREMs) are also evaluated when available as the basis for recommendations and/or discussion in these guidelines.

Evidence tables summarizing key information from relevant studies are generated to facilitate the formulation of recommendations, to enhance comprehension of recommendations after publication, and to

Table 1 Classes of recommendations

Classes of recommendations	Definition	Wording to use
Class I	Evidence and/or general agreement that a given treatment or procedure is beneficial, useful, effective.	Is recommended or is indicated
Class II	Conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of the given treatment or procedure.	
Class IIa	Weight of evidence/opinion is in favour of usefulness/efficacy.	Should be considered
Class IIb	Usefulness/efficacy is less well established by evidence/opinion.	May be considered
Class III	Evidence or general agreement that the given treatment or procedure is not useful/effective, and in some cases may be harmful.	Is not recommended

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Table 2 **Levels of evidence**

Level of evidence A	Data derived from multiple randomized clinical trials or meta-analyses.
Level of evidence B	Data derived from a single randomized clinical trial or large non-randomized studies.
Level of evidence C	Consensus of opinion of the experts and/or small studies, retrospective studies, registries.

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reinforce transparency in the guidelines' development process. The tables are published in their own section of ESC Guidelines and reference specific recommendation tables.

After an iterative process of deliberations, a first Task Force vote on all recommendations is conducted prior to the initiation of rounds of review. A second Task Force vote on all recommendations is conducted after the final round of review and revision. For each vote, the Task Force follows ESC voting procedures and all recommendations require at least 75% agreement among voting members to be approved. Voting restrictions may be applied based on declarations of interests.

The writing and reviewing panels provide declaration of interest forms for all relationships that might be perceived as real or potential sources of conflicts of interest. Their declarations of interest are reviewed according to the ESC declaration of interest rules, which can be found on the ESC website (http://www.escardio.org/doi) and are compiled in a report published in a supplementary document with the guidelines. Funding for the development of ESC Guidelines is derived entirely from the ESC with no involvement of the healthcare industry.

The CPG Committee supervises and coordinates the preparation of new guidelines and approves their publication. In addition to review by the CPG Committee, ESC Guidelines undergo multiple rounds of double-blind peer review on a dedicated online review platform. The review is conducted by topic experts, including members from ESC National Cardiac Societies and from relevant ESC Subspecialty Communities. Guideline Task Forces consider all review comments and are required to respond to all those classified as major. After appropriate revisions, the Task Force and the CPG Committee members approve the final document for publication in the *European Heart Journal*.

Unless otherwise stated, ESC Guidelines content refers to sex, understood as the biological condition of being male or female, defined by genes, hormones, and sexual organs. Off-label use of medication may be presented in this guideline if a sufficient level of evidence shows that it can be considered medically appropriate for a given condition. However, decisions on off-label use must be made by the responsible health professional giving special consideration to ethical rules concerning healthcare, the specific situation of the patient, patient consent, and country-specific health regulations.

2. Introduction

2.1. Why we need new Guidelines on cardiovascular disease and pregnancy

Since the previous version of these Guidelines was published in 2018, new evidence has emerged, and clinical focus has changed in various aspects, requiring an updated discussion. These include the importance of the Pregnancy Heart Team, the modalities for pre-pregnancy counselling and risk stratification, and drugs during pregnancy, lactation and/or breastfeeding, and post-partum stages. In *Table 3*, the most relevant updates are listed with their respective rationale.

2.2. Why these Guidelines are important

Cardiovascular disease (CVD) is a major cause of maternal mortality and morbidity. With these new Guidelines we wish to provide updated evidence-based guidance for patients and caregivers. Reducing maternal mortality and morbidity is a key priority of the World Health Organization (WHO).

In managing maternal and foetal health, it is essential to balance the risks and benefits of maternal and foetal therapeutic needs. Due to the scarcity of prospective or randomized studies within this field, which often cannot be performed for ethical reasons, most recommendations in these Guidelines are based on evidence level C. Consequently, there is an ongoing need for more registries, such as the Registry of Pregnancy and Cardiac Disease (ROPAC) and the European Surveillance of Congenital Anomalies network, and prospective studies to enhance our understanding in this area.

2.3. What is new

As mentioned in *Section 2.1*, this new version of the guidelines not only includes an update to several recommendations (Table 4) but also introduces a structural revision. See Supplementary data online (*Table S1*) for a detailed overview of the new recommendations.

Below we discuss the key updates per section, highlighting a selection of new and revised recommendations along with their rationale.

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Table 3 Updates of the 2025 Guidelines on cardiovascular disease and pregnancy

Topic	New information	Rationale
Pregnancy Heart Team[a]	Broader acceptance, dedicated section	Ensure comprehensive care throughout reproductive stages[b]
Risk stratification[a,c]	mWHO 2.0 classification, refined and expanded clinical categories	More data have emerged, necessitating more nuanced risk assessment for patient counselling[1,2]
Clinical data and research	ROPAC[3–12] and PPCM[13–17] registries Cardiomyopathies Primary arrhythmia syndromes	New or updated clinical management
Clinical scenarios	Algorithms for management of clinical situations in pregnant women	Provide practical information for the clinical cardiologist
Genetic testing and counselling	Advancements in testing and pre-implantation procedures	Incorporation of latest management of genetic testing and counselling
Revision of contraindications (COR III) for pregnancy in women classified as mWHO class IV	Emphasis on the critical role of comprehensive counselling by the Pregnancy Heart Team (COR I)	Recognition of a woman's autonomy in making reproductive choices Promoting a detailed and transparent dialogue about the heightened risks and encouraging shared decision-making
Adverse pregnancy outcomes (APO)	Increased focus on long-term outcomes	Evidence supports the need for thorough discussion and management of APOs[18]

COR, class of recommendation; mWHO, modified World Health Organization; PPCM, peripartum cardiomyopathy; ROPAC, Registry of Pregnancy and Cardiac Disease.
[a]See *Section 4*.
[b]See *Figure 2*.
[c]See *Table 6*.

Table 4 New recommendations

Recommendations	Class[a]	Level[b]
***Section 4*. The Pregnancy Heart Team**		
Although the concept of the Pregnancy Heart Team was previously part of the general principles, it has now been given its own dedicated section, which covers all aspects from pre-conception through to the postpartum period.		
A discussion by the Pregnancy Heart Team about the high risk of maternal mortality or morbidity and the related high foetal risk is recommended for women with mWHO 2.0 class IV conditions, including a shared decision-making process for pregnancy termination, involving psychological support.	I	C
It is recommended that women with CVD of mWHO 2.0 class II–III and above are evaluated and managed by a Pregnancy Heart Team from pre-pregnancy onwards through pregnancy and post-partum.	I	C
Measurement of BNP and NT-proBNP levels should be considered prior to pregnancy in women with HF of any aetiology, including previous PPCM, cardiomyopathy, ACHD, and PAH, and be monitored during pregnancy according to the underlying disorder and in case of new-onset or worsening symptoms.	IIa	B
***Section 5*. Drugs during pregnancy and lactation**		
Given the importance of medication use throughout this document, this section has been brought forward and revised. The former comprehensive medication table has been moved to the Supplementary data online, and we provide a summary figure (Figure 6) listing the (contra)indicated medications.		
***Section 6*. Pregnancy in women with cardiomyopathies and primary arrhythmia syndromes**		
This section has been expanded since 2018 for advice in specific cardiomyopathies and primary arrhythmias.		
Vaginal delivery is recommended in most women with CMPs, unless there are obstetric indications for caesarean section, severe HF (EF <30% and/or NYHA class III/IV), uncontrolled arrhythmias, or severe outflow obstruction (≥50 mmHg) in women with HCM, or in women presenting in labour on VKAs.	I	C
In women with DCM and worsening of EF during pregnancy, counselling on the risk of recurrence during a subsequent pregnancy is recommended in all cases, even after recovery of LV function.	I	C
It is recommended that women with HCM with symptomatic LV dysfunction (EF <50%) and or severe LVOTO (≥50 mmHg) wishing to become pregnant are counselled by the Pregnancy Heart Team regarding the high risk of pregnancy-related adverse events.	I	C
Myosin inhibitors are not recommended in women during pregnancy due to lack of safety data.	III	C

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Beta-blockers, with pre-pregnancy dose and with nadolol and propranolol as drugs of choice, are recommended during pregnancy in women with LQTS.	I	B
It is recommended to continue beta-blocker therapy during lactation in women with LQTS to reduce arrhythmic risk.	I	B
Pre-pregnancy dose beta-blockers of nadolol or propranolol is recommended in patients with LQT2, particularly in the post-partum period, which represents a high-risk period for life-threatening arrhythmias.	I	B
Beta-blockers, with pre-pregnancy dose and with nadolol and propranolol as drugs of choice, are recommended during pregnancy and lactation in women with CPVT.	I	C
Flecainide, in addition to beta-blockers, is recommended in women with CPVT who experience cardiac events, such as syncope, VT, or cardiac arrest during pregnancy.	I	C
***Section 7*. Peripartum cardiomyopathy**		
We have provided a separate section on PPCM in these guidelines.		
Genetic counselling and testing should be considered in women with PPCM.	IIa	C
When a reversible course of HF is assumed, treatment in accordance with HF guidelines should be considered for at least 12 months after complete LV recovery (normalization of LV volumes and EF).	IIa	C
***Section 8*. Pregnancy in women with aortopathies**		
Since 2018, significant evidence has emerged in the context of heritable thoracic aortic disease (HTAD), supporting a more gene- and variant-based approach, which has been incorporated in this version of the guidelines.		
It is recommended that women with a history of aortic dissection or -surgery have pre-pregnancy counselling about the high risk by an extended Pregnancy Heart Team considering the presence and type of genetic variant, aortic morphology, growth rate, and aetiology of aortic dissection.	I	C
***Section 9*. Pregnancy in women with known congenital heart disease**		
This section has undergone a major update based on recent reports, which have been summarized in a clear and concise table.		
It is recommended that all women with Fontan circulation who wish to become pregnant receive counselling from the Pregnancy Heart Team regarding the high risk of pregnancy-related adverse events.	I	C
***Section 10*. Pregnancy in women with pulmonary arterial hypertension**		
This subject is now covered in a separate section in these guidelines, in line with growing insights into management.		
It is recommended that women of childbearing potential with PAH wishing to become pregnant are counselled by a multidisciplinary team regarding the very high risk of pregnancy-related adverse events, encouraging a shared decision-making process about whether to become pregnant.	I	C
***Section 11*. Venous thromboembolism in pregnancy and post-partum**		
Guidance on the involvement of an expert team and more prompt initiation of treatment are now provided in dedicated flowcharts and recommendations.		
In pregnant women or women in the post-partum period with suspicion of venous thromboembolism (VTE) [deep vein thrombosis (DVT) and/or PE], an immediate formal diagnostic assessment with validated methods is recommended and should not be postponed.	I	B
***Section 12*. Pregnancy in women with acquired heart disease**		
Recommendations for emergency situations are provided for acquired heart diseases in addition to the new sections on cardio-oncology and heart transplantation.		
Recommendations for coronary artery disease and pregnancy		
In pregnant women with chest pain, it is recommended to exclude life-threatening cardiovascular conditions, including PE, ACS (including SCAD), and acute aortic syndrome.	I	C
The duration of DAPT (aspirin and clopidogrel) in pregnant women undergoing coronary stent implantation is recommended to be the same as in non-pregnant women, with an individual approach considering ischaemic risk and delivery-related bleeding risks.	I	C
Continuation of statins may be considered during pregnancy in women with established ASCVD.	IIb	C
Recommendations for hypertensive disorders and pregnancy		
It is recommended to aim for systolic BP <140 mmHg and diastolic BP <90 mmHg in pregnant women.	I	B
In severe hypertension, drug treatment with i.v. labetalol, urapidil, nicardipine, or oral short acting nifedipine or methyldopa is recommended for acute reduction in blood pressure. Intravenous hydralazine is a second-line option.	I	C
Recommendations for supraventricular tachycardia and pregnancy		
Therapeutic anticoagulation with LMWH is recommended for pregnant women with persistent or permanent AF at elevated thromboembolic risk.	I	C
Flecainide, in addition to beta-blockers, should be considered for long-term AF rhythm control in pregnancy.	IIa	C
Recommendation for ventricular tachycardia, device implantation and catheter ablation and pregnancy		
When performing catheter ablation during pregnancy, the use of non-fluoroscopic mapping and navigation systems should be considered.	IIa	C

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Recommendations for cardiac arrest and pregnancy		
Continuous manual left uterine displacement during CPR in pregnant women (≥20 weeks) with cardiac arrest is recommended to relieve aortocaval compression.	I	C
It is recommended to establish i.v. access above the diaphragm to ensure that the i.v. therapy is not obstructed by the gravid uterus.	I	C
It is recommended that no drugs are withheld in pregnant women with cardiac arrest due to concerns of teratogenicity.	I	C
Recommendation for congenital atrioventricular block and pregnancy		
In pregnant women with asymptomatic congenital AV block, normal cardiac anatomy and function, a narrow QRS complex, and ventricular rate (≥50 b.p.m.) a prophylactic temporary pacemaker during delivery is not recommended.	III	C
Recommendation for native valve disease and pregnancy		
Valve surgery during pregnancy should only be considered when there is a maternal mortality risk and other treatment options have failed.	IIa	C
Recommendation for prosthetic valves disease and pregnancy		
It is recommended that a care plan documenting the agreed anticoagulant strategy (including the decision to continue VKAs or converting to therapeutic-dose LMWH in the first trimester) is in place for women of childbearing age with a MHV prior to pregnancy or as soon as pregnancy is recognized.	I	C
Recommendations for chronic and acute heart failure and pregnancy		
Inotropes and/or vasopressors are recommended in pregnant women with cardiogenic shock with levosimendan, dobutamine, and milrinone as recommended agents.	I	C
ACE-Is, ARBs, ARNIs, MRAs, ivabradine, and SGLT2 inhibitors are not recommended during pregnancy due to adverse foetal effects.	III	C
Recommendations for heart transplantation and pregnancy		
It is recommended to postpone pregnancy until at least 1 year after heart transplantation, taking individual risk factors into account.	I	C
In women with a heart transplant, it is recommended that immunosuppression serum drug levels are monitored during pregnancy every 4 weeks until the 32nd week, then every 2 weeks until the 36th week, then weekly until delivery and for 6–12 months after delivery to guide dosing.	I	C
Recommendation for cardio-oncology and pregnancy		
It is recommended that pregnant women with cancer who require cardiotoxic cancer therapy are jointly managed by the Pregnancy Heart Team and the cardio-oncology team.	I	C
***Section 13*. Long-term effects of adverse pregnancy outcomes**		
This is a completely new section in the guidelines, reflecting the growing recognition of the importance of APOs.		
It is recommended to undertake a cardiovascular risk assessment in women with APOs, to recognize and document APOs when CVD risk is evaluated in women, and to provide counselling on the importance of healthy lifestyle choices that optimize cardiovascular health.	I	B

ACE-I, angiotensin-converting enzyme inhibitor; ACHD, adult congenital heart disease; ACS, acute coronary syndrome; AF, atrial fibrillation; APO, adverse pregnancy outcomes; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor/neprilysin inhibitor; ASCVD, atherosclerotic cardiovascular disease; AV, atrioventricular; BNP, brain natriuretic peptide; BP, blood pressure; CMP; cardiomyopathy; CPR, cardiopulmonary resuscitation; CPVT, catecholaminergic polymorphic ventricular tachycardia; CVD, cardiovascular disease; DAPT, dual antiplatelet therapy; DCM, dilated cardiomyopathy; DVT, deep vein thrombosis; EF, ejection fraction; HCM, hypertrophic cardiomyopathy; HF, heart failure; HTAD, heritable thoracic aortic disease; i.v., intravenous; LMWH, low-molecular-weight heparin; LQT2, long QT syndrome type 2; LQTS, long QT syndrome; LV, left ventricle; LVOTO, left ventricular outflow tract obstruction; MHV, mechanical heart valve; MRA, mineralocorticoid receptor antagonist; mWHO, modified World Health Organization; NT-proBNP, N-terminal pro-brain natriuretic peptide; NYHA, New York Heart Association; PAH, pulmonary arterial hypertension; PE, pulmonary embolism; PPCM, peripartum cardiomyopathy; SCAD, spontaneous coronary artery dissection; SGLT2, sodium–glucose co-transporter-2; VKA, vitamin K antagonist; VT, ventricular tachycardia; VTE, venous thromboembolism.
[a]Class of recommendation.
[b]Level of evidence.

2.4. What has changed

Table 5 lists the recommendations with a revised Class of Recommendation since the 2018 Guidelines for the management of cardiovascular diseases during pregnancy. See Supplementary data online (*Table S2*) for a detailed overview of all modified recommendations.

Table 5 Revised recommendations

Recommendations in 2018	Class[a]	Level[b]	Recommendations in 2025	Class[a]	Level[b]
***Section 4*. The Pregnancy Heart Team**					
Prophylactic antibiotic therapy to prevent endocarditis during delivery is not recommended.	III	C	Systemic antibiotic prophylaxis may be considered for delivery in women at high risk.	IIb	C
***Section 6*. Recommendations for cardiomyopathies and pregnancy**					
In patients with HCM, it is recommended that beta-blockers are continued in women who used them before pregnancy.	I	C	Continuation of beta-blockers should be considered during pregnancy in women with CMPs, with close follow-up of foetal growth.	IIa	C

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***Section 8*. Recommendations for aortopathies, cardiac surgery, and pregnancy**					
Pregnancy is not recommended in patients with vascular Ehlers–Danlos syndrome.	III	C	It is recommended that women with vascular Ehlers–Danlos syndrome wishing to become pregnant are counselled regarding the very high risk of pregnancy-related adverse events by a multidisciplinary team, considering family history, genetic variant, and previous vascular events.	I	C
Beta-blocker therapy throughout pregnancy should be considered in women with Marfan syndrome and other heritable thoracic aortic diseases.	IIa	C	Beta-blocker therapy throughout pregnancy and in the post-partum period is recommended in women with MFS and other HTADs.	I	C
***Section 9*. Recommendations for congenital heart disease and pregnancy**					
Patients with a systemic right ventricle (Mustard/Senning or congenitally corrected TGA), in NYHA class III/IV, systemic ventricular dysfunction (EF <40%), or severe TR should be advised against pregnancy.	IIa	C	It is recommended that women with a systemic RV (Mustard/Senning or congenitally corrected TGA), in NYHA class III/IV, systemic ventricular dysfunction (EF <40%), or severe TR wishing to become pregnant are counselled by the Pregnancy Heart Team regarding the high risk of pregnancy-related adverse events.	I	C
***Section 12*. Recommendations for acquired heart disease and pregnancy**					
An invasive management strategy should be considered for NSTE ACS with high-risk criteria.	IIa	C	It is recommended to manage pregnant women with ACS in the same way as non-pregnant women, including diagnostic investigations and interventions.	I	C
Catheter ablation with electro-anatomical systems should be considered in experienced centres in cases of drug-refractory and poorly tolerated SVT.	IIa	C	Catheter ablation may be considered in pregnant women with recurrent, long symptomatic SVT or with contraindications to pharmacological therapies.	IIb	C
Balloon aortic valvuloplasty should be considered during pregnancy in patients with severe aortic stenosis and severe symptoms.	IIa	C	In very selected symptomatic pregnant women with severe aortic stenosis not responding to medical therapy, non-surgical options such as balloon valvuloplasty or TAVI may be considered.	IIb	C
A bioprosthesis should be considered in young women contemplating pregnancy.	IIa	C	A bioprosthetic valve is recommended (over a mechanical valve) in young women contemplating pregnancy requiring a valve prosthesis.	I	B
During the second and third trimesters, LMWH with anti-factor Xa level monitoring and dose adjustment (see separate recommendations) may be considered in women who need a high dose of VKA after patient information and consent.	IIb	C	During the second and third trimesters until the 36th week, continuing VKAs should be considered in women with prosthetic heart valves at higher risk of thrombosis.	IIa	C

ACS, acute coronary syndrome; CMP, cardiomyopathy; EF, ejection fraction; HCM, hypertrophic cardiomyopathy; HTAD, heritable thoracic aortic disease; LMWH, low-molecular-weight heparin; MFS, Marfan syndrome; NSTE ACS, non-ST-elevation myocardial infarction; NYHA, New York Heart Association; RV, right ventricle; SVT, supraventricular tachycardia; TAVI, transcatheter aortic valve implantation; TGA, transposition of the great arteries; TR, tricuspid regurgitation; VKA, vitamin K antagonist.
[a]Class of recommendation.
[b]Level of evidence.

3. General considerations

3.1. Epidemiology

Within high-income countries the number of pregnancies and deliveries in women with acquired, congenital, or inherited CVD is growing significantly.[19–21] This trend originates from several factors: higher maternal age at first pregnancy, a growing number of women with congenital heart disease reaching childbearing age, and a rising prevalence of cardiovascular comorbidities.[19–21] Globally, up to 4% of pregnancies are complicated by CVD, rising to 10% when including hypertensive disorders.[22–25] Maternal CVD is now the leading cause of non-obstetric mortality in pregnant women[24,26], accounting for 33% of pregnancy-related deaths worldwide.[25,27,28] In women with pre-existing CVD, up to 16% of pregnancies are complicated by CVD.[29–32] Notably, 68% of pregnancy-related deaths caused by CVD are preventable.[33] Women with CVD during pregnancy face higher risk of cardiac events later in life, making secondary prevention crucial.[25] Adverse neonatal outcomes occur in ~25% of these pregnancies in women with CVD[34,35] with high rates of obstetric complications (17%) and maternal mortality/morbidity (11%).[34,35] Of note, recent research indicates that pre-existing CVD in the mother is associated with an increased risk of CVD in her children, and this association is unlikely to be explained by unmeasured familial or genetic factors.[36]

3.2. Physiology of pregnancy

Pregnancy induces physiological changes in the cardiovascular system to meet the increased metabolic needs of the mother and foetus (*Figure 1*). These changes occur from the early stages of pregnancy onward. Starting at 6 weeks of gestation, stroke volume and cardiac output (CO) increase

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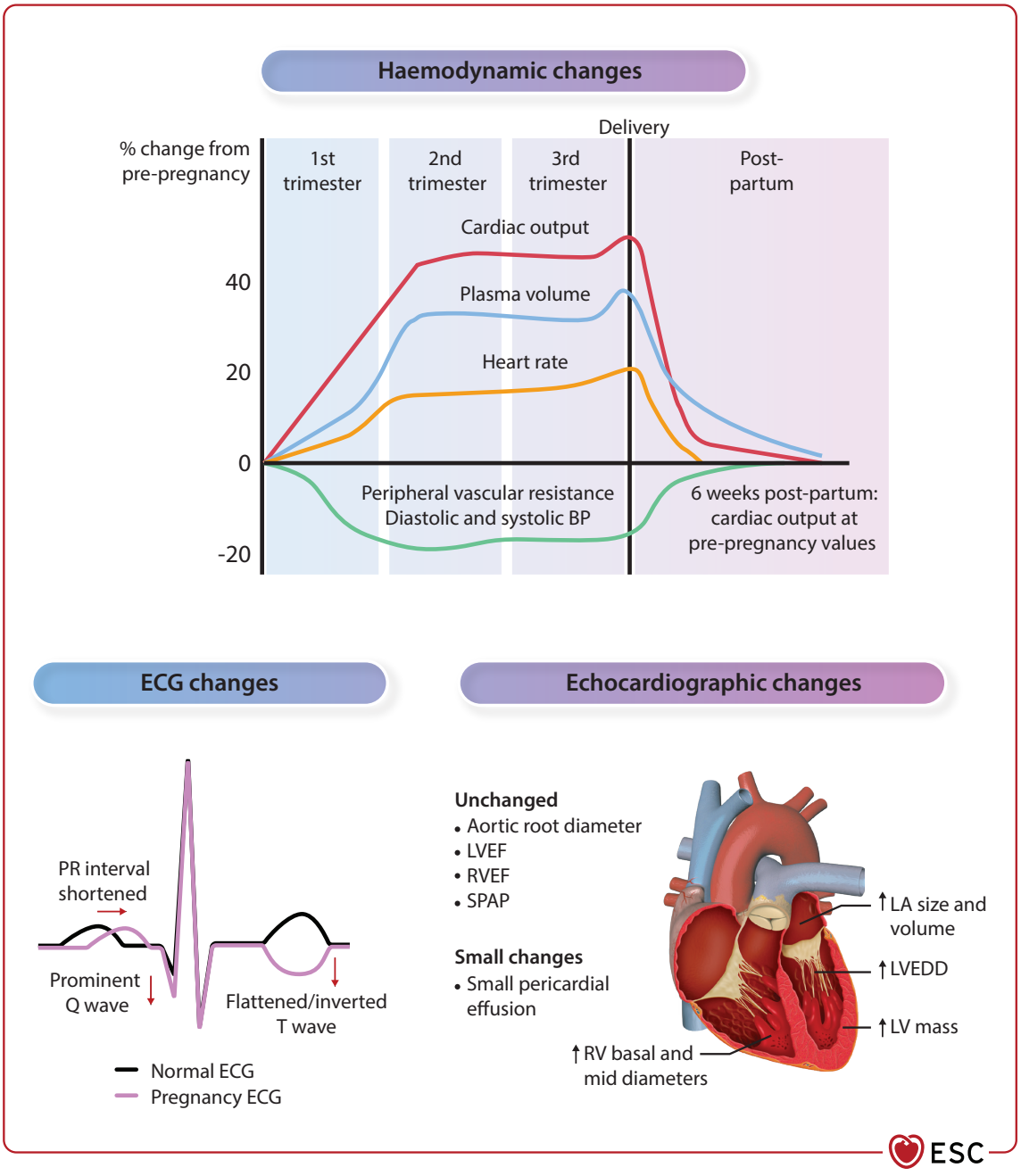

Figure 1 Physiology of haemodynamic changes, and changes in electrocardiogram and echocardiography during and post pregnancy. BP, blood pressure; ECG, electrocardiogram; LA, left atrium; LV, left ventricle; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; RV, right ventricle; RVEF, right ventricular ejection fraction; SPAP, systolic pulmonary artery pressure.

by 30%–50%, and heart rate increases by 10–20 beats per minute. Peripheral vascular resistance decreases by 20%–50%.[37]

Left and right atrial and ventricular diameters and volumes increase while ventricular function is preserved.[38] Blood pressure and CO increase during labour.[39] After delivery, the uterus contracts, and CO drops rapidly to ~15%–25% above normal. A gradual decrease of CO follows over the next 3–4 weeks and reaches pre-pregnancy levels at ~6 weeks post-partum.

In women with heart disease, left and right ventricular adaptation to pregnancy can be suboptimal and can lead to heart failure (HF) and atrial and ventricular tachyarrhythmias. Atrial arrhythmias may develop in response to cardiac stretch and hormonal changes in pregnancy and may not be well tolerated in women with CVD. The haemodynamic and hormonal changes during pregnancy are risk factors for aortic dissection in women with aortopathy.[40] Pregnancy is a hypercoagulable state associated with an increased risk of thromboembolism.[41] Increased activity of gastrointestinal–hepatic metabolism, liver enzyme systems, glomerular filtration rate and plasma volume, changes in protein binding, and decreased serum albumin levels all contribute to changes in the pharmacokinetics of many drugs.[42]

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4. The Pregnancy Heart Team

4.1. Concept and requirements

The central role of the Pregnancy Heart Team is illustrated in Figure 2. The concept of the Pregnancy Heart Team was first introduced in the 2018 ESC Guidelines for the management of cardiovascular diseases during pregnancy.[43] It has become an established component in the care of women with CVD or those who develop cardiovascular problems during pregnancy.[44–49] This care provision commences prior to pregnancy and persists through the postpartum period. Research has shown that management by a Pregnancy Heart Team is associated with favourable maternal, foetal, and healthcare services outcomes, including lowered maternal mortality and readmission rates and subsequently improved patient safety.[50] Institutional Pregnancy Heart Teams should be established in referral hospitals, taking into account the geographical regions, disciplines represented, and numbers of births, as well as sociocultural aspects.[44,45,47–49] Maintaining a balance between the need for follow-up by such teams and the workload on these teams is crucial, emphasizing the importance of carefully selecting women who should be directed to a Pregnancy Heart Team. Patient selection is best accomplished by a risk assessment using the modified World Health Organization (mWHO) 2.0 classification (*Table 6*). The Pregnancy Heart Team should encompass a core team that can be expanded with other experts (see *Figure 3*), tailored to women's physical and mental health or emerging complications.[53]

The primary responsibilities of the Pregnancy Heart Team encompass risk assessment, collaborative care plan development, continuous progress monitoring, coordination, patient education, and psychological counselling. A staged approach is recommended, from pre-conception and pregnancy through to labour, delivery, and post-partum care. Promoting shared decision-making is essential at all stages. Not every hospital needs to establish a dedicated Pregnancy Heart Team, but each hospital should establish communication and collaboration with nearby expert teams, optimizing emergency and elective referral pathways within a shared-approach care model.

4.2. Pre-pregnancy counselling and family planning

Women should receive pre-pregnancy counselling and education about the maternal, foetal, and transmission risk(s).[54] In adolescents diagnosed with congenital or inherited heart disease, tailored discussions about reproductive health should start early, ideally from menarche. Girls and women with congenital heart disease may not demonstrate appropriate understanding of safe contraception and pregnancy risk. High rates of unintended pregnancies (up to 45%) have been reported in adolescents with congenital heart disease.[55] Transition programmes showed improved disease-related knowledge levels in adolescents with congenital heart disease.[56–58]

4.2.1. Risk assessment

A personalized pregnancy-related risk assessment is needed in all women with CVD and should encompass the specific cardiovascular diagnosis, functional status, and medication regimen, as well as non-cardiac risk factors such as maternal age, smoking history, comorbidities, body mass index (BMI), obstetric history, logistical care aspects, maternal ethnicity, and socioeconomic status. It is also crucial to integrate foetal and obstetric outcomes with specific cardiovascular considerations. Maternal preferences should be thoroughly explored as part of the shared decision-making process.

4.2.1.1. Maternal risk assessment

Depending on the diagnosis, a cardiovascular assessment may include imaging, biomarker-level assessment, and functional testing. Cardiopulmonary exercise testing can be useful for pre-pregnancy risk stratification.[59,60] Assessment may include re-evaluation after discontinuation of teratogenic medications and whether pre-pregnancy intervention is required.

Various scoring systems are available to assess maternal risk and foetal risk. These do not fully explore the interaction with non-cardiac risk factors, only focus on maternal cardiac events, and have mainly been validated in higher-income countries. Further adaptation and validation in other countries may be required.[51] Despite these limitations, disease-specific risks can be effectively assessed using the mWHO classification, validated as the best-available risk assessment model.[1,61] However, the mWHO classification is oriented towards adult congenital heart disease (ACHD) and identifying those at the highest risk. Therefore, the mWHO 2.0 classification has now been expanded with other CVDs and refined by integrating the Cardiac Disease in Pregnancy study (CARPREG) II (*Table 6*).[52] Specific expertise and collaborative management by a Pregnancy Heart Team is mandatory for all women with a condition of mWHO 2.0 class II–III or above.

4.2.2. Genetic counselling

Several CVDs have a genetic basis, including heritable cardiac conditions such as some aortopathies, channelopathies, cardiomyopathies, congenital heart disease, and subsets of pulmonary arterial hypertension (PAH) and thromboembolic disease. Most show autosomal dominant inheritance with a 50% transmission risk. Knowledge of an underlying pathogenic/likely pathogenic (P/LP) variant (adjusted terminology for mutation) is of increasing importance to better assess pregnancy-related outcomes and to adjust management (*Figure 4*).[62,63] Therefore, it is recommended that genetic testing for CVD is performed pre-pregnancy in a specialized cardiogenetic centre or a network model with access to a multidisciplinary team, involving appropriately trained professionals with expertise in genetic testing methodology, sequence variant interpretation, clinical application of genetic testing, and pre- and post-testing genetic counselling about the transmission risk and the variable expression of an inherited genetic condition.[64,65]

4.2.2.1. Pre-natal and pre-implantation diagnosis

A timely discussion about pre-implantation (or pre-gestational) genetic testing and pre-natal testing should be offered to every woman and/or couple when there is a known parental monogenic or chromosomal abnormality. Pre-implantation genetic testing requires *in vitro* fertilization (IVF). The modalities of pre-natal and pre-implantation testing, including precautions, are summarized in *Table 7*. The decision whether to pursue pre-implantation or pre-natal genetic testing should include consideration of a spectrum of aspects related to the disease, including cultural, religious, and legal issues, but also accessibility of required techniques and expertise.[67] Counselling should be provided at an experienced centre with an expert multidisciplinary team. An individualized approach is required to ensure autonomous choice and informed decision-making within the local ethical and legal framework. Several of these options take time and require early referral.

4.2.3. Reproductive technology

Infertility rates in most women with CVD are similar to those in the general population, but managing infertility and medically assisted reproductive treatment is more complex.[68]

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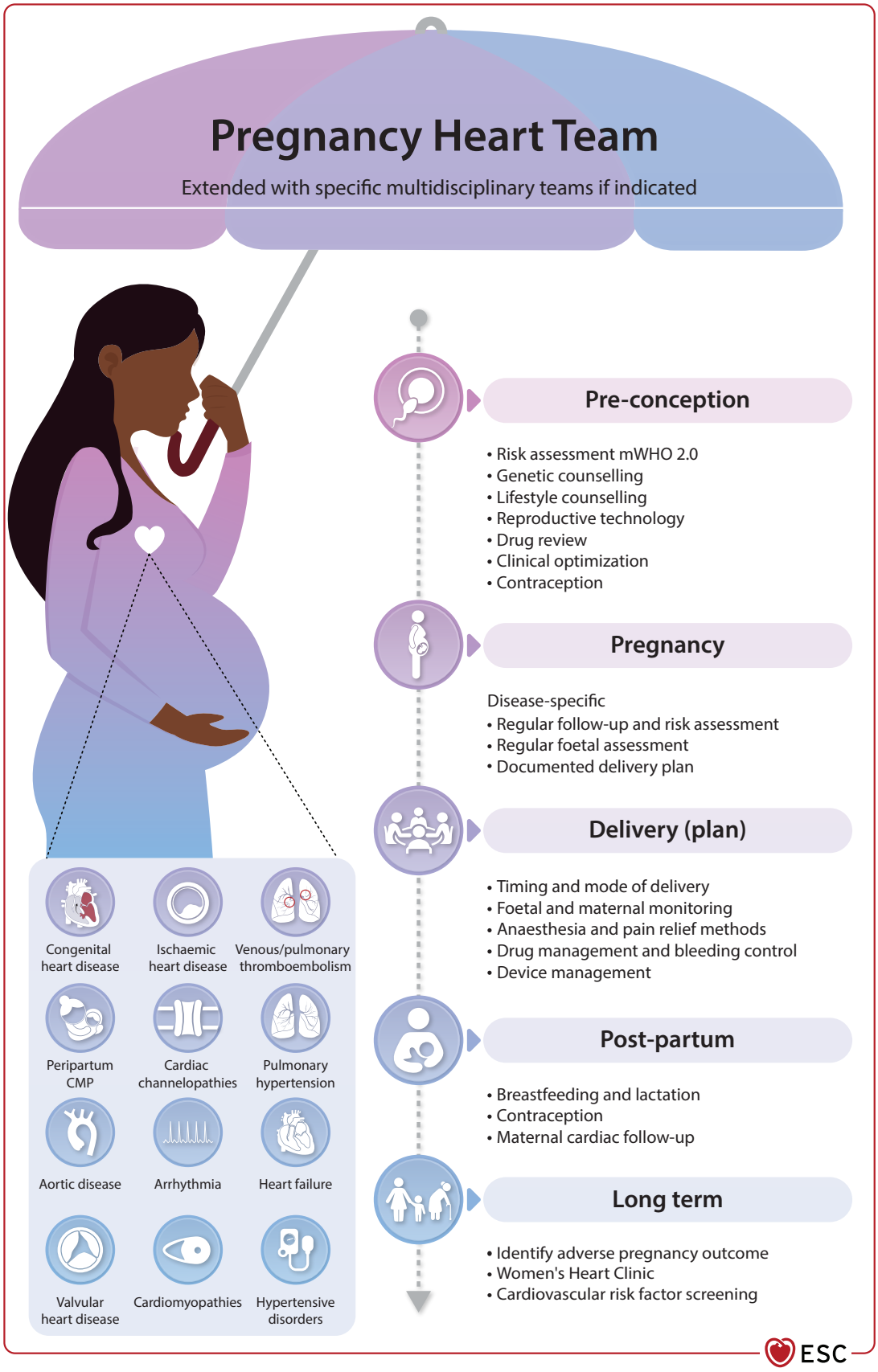

Figure 2 Central illustration. Role of Pregnancy Heart Team in pregnancy pathway. CMP, cardiomyopathy; mWHO, modified World Health Organization.

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Table 6 Modified World Health Organization 2.0 classification of maternal cardiovascular risk

	mWHO 2.0 I	mWHO 2.0 II	mWHO 2.0 II–III	mWHO 2.0 III	mWHO 2.0 IV
Diagnosis	**Ventricular (dys)function + pulmonary hypertension**				
			Mild left ventricular impairment: EF >45%. Significantly impaired RV (subpulmonary) function.	Moderate left ventricular impairment: EF 30%–45%. Previous PPCM with not more than mild residual left ventricular impairment.	Severe left ventricular impairment: EF <30% or NYHA class III/IV. Previous PPCM with more than mild left ventricular impairment. PAH.
	Arrhythmias				
	Atrial or ventricular ectopic beats, isolated.	Most supraventricular arrhythmias. Bradycardia requiring pacemaker.	Low-risk LQTS: no previous events + on full dose beta-blocker therapy. Low-risk CPVT: well controlled by medical therapy. BrS with no previous events.	Sustained ventricular tachycardia from any aetiology. LQT2 (post-partum). Symptomatic CPVT and LQTS not adequately controlled by therapy. BrS with previous events.	
	Cardiomyopathy				
	HCM: genotype-positive + phenotype-negative.		Low-risk ARVC: genotype-positive + no or mild phenotype. HCM without complications. DCM/NDLVC with normal or mild left ventricular impairment: EF >45%.	ARVC with moderate/severe disease. HCM with arrhythmic and/or moderate haemodynamic complications. DCM/NDLVC with moderate left ventricular impairment: EF 30%–45%.	DCM/NDLVC with severe left ventricular impairment: EF <30% or NYHA class III/IV. HCM with symptomatic severe outflow tract obstruction: ≥50 mmHg. HCM with severely symptomatic LV dysfunction (EF <50%).
	Congenital heart disease				
	Successfully repaired simple lesions without significant residual (haemodynamic) complications (atrial or ventricular septal defect, patent ductus arteriosus, anomalous pulmonary venous drainage).	Unoperated uncomplicated atrial or ventricular septal defect. Repaired tetralogy of Fallot without significant residual haemodynamic/arrhythmic lesions. Transposition of the great arteries with arterial switch without significant residual lesions.	Repaired atrioventricular septal defect without significant residual lesions. Uncomplicated Ebstein anomaly: mild to moderate TR, no tricuspid stenosis, no accessory pathway.	Unrepaired cyanotic heart disease (not Eisenmenger). Systemic RV with good or mildly decreased ventricular function. Uncomplicated Fontan circulation: good ventricular function, no significant valve disease or arrhythmias, good exercise tolerance, and normal arterial saturations. Ebstein anomaly with any complication.	Systemic RV with moderate or severely decreased ventricular function. Fontan with any complication. Eisenmenger syndrome.

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		mWHO 2.0 I	mWHO 2.0 II	mWHO 2.0 II–III	mWHO 2.0 III	mWHO 2.0 IV
		Valvular heart disease				
		Small or mild • pulmonary stenosis • mitral valve prolapse without significant regurgitation.		Native, homograft or tissue valve disease not considered mWHO 2.0 I or IV: mild mitral stenosis, moderate aortic stenosis. Moderate valvular regurgitation.	Uncomplicated mechanical valve with stable well controlled INRs. Moderate mitral stenosis. Severe asymptomatic aortic stenosis. Severe left-sided valvular regurgitation.	Severe mitral stenosis. Severe symptomatic aortic stenosis.
		Aortopathy				
		Non-HTAD mild aortic dilatation (<40 mm).	Turner syndrome without cardiovascular features (BAV, coarctation, AHT, aortic dilatation).	Marfan or other HTAD syndrome without aortic dilatation. Aorta <45 mm in BAV pathology. Repaired coarctation.	Moderate aortic dilatation: 40–45 mm in Marfan syndrome or other HTAD; 45–50 mm in BAV, Turner syndrome ASI 20–25 mm/m^2, other aortic dilatation <50 mm. Marfan with previous aortic root replacement. Previous aortic dissection with stable diameter.	Severe aortic dilatation: >45 mm in Marfan syndrome or other HTAD, >50 mm in BAV, ASI >25 mm/m^2 in Turner syndrome, other aortic dilatation >50 mm. Vascular Ehlers–Danlos syndrome. Severe (re)coarctation. Previous aortic dissection with increasing diameter.
		Acquired + coronary heart disease + other				
					Prior SCAD. Prior ischaemic cardiac event (STEMI/NSTE ACS). Prior adverse pregnancy outcome requiring hospitalization. Prior adverse cardiovascular effects of cancer treatment.	
Risk		No detectable increased risk of maternal mortality and no/mild increased risk in morbidity.	Small increased risk of maternal mortality or moderate increase in morbidity.	Intermediate increased risk of maternal mortality or moderate to severe increase in morbidity.	Significantly increased risk of maternal mortality or severe morbidity.	Extremely high risk of maternal mortality or severe morbidity.
Average maternal cardiac event rates[a]	Van Hagen et al. (2016)[51]	9.9%	7.7%	17.7%	28.9%	50.3%
	Silversides et al. (2018)[52]	3.1%	21.7%	12.8%	21.1%	35.6%
Individualize each maternal risk with the modifiers below[b] (derived from CARPREG II)[52]						
CARPREG II score: 1 point • No prior cardiac intervention indicated • Late pregnancy assessment			**CARPREG II score: 2 points** • Ventricular dysfunction • High-risk left-sided valve disease or outflow tract obstruction • Pulmonary hypertension • Coronary artery disease • High-risk aortopathy		**CARPREG II score: 3 points** • Prior cardiac event or arrhythmias • Baseline NYHA III/IV or cyanosis • Mechanical valve	

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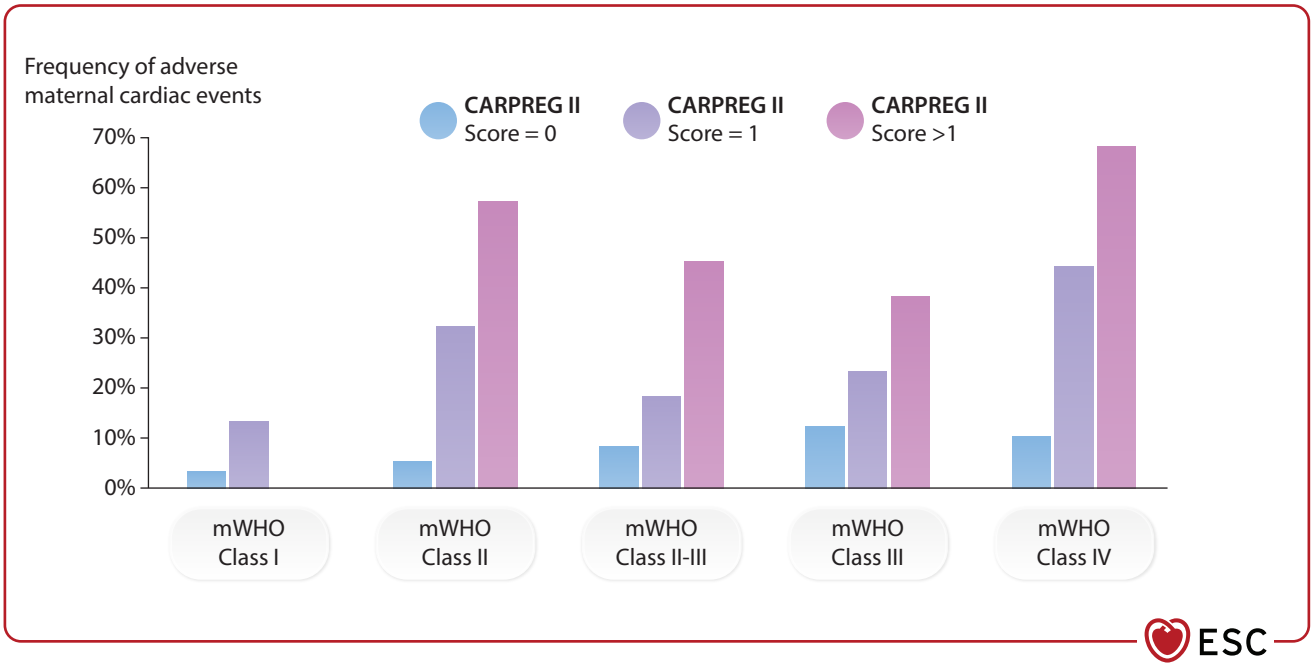
	mWHO 2.0 I	mWHO 2.0 II	mWHO 2.0 II–III	mWHO 2.0 III	mWHO 2.0 IV
Involvement of the Pregnancy Heart Team	No	No	Yes	Yes	Yes
Counselling	Yes (by regular healthcare professional)	Yes (by regular healthcare professional)	Yes: expert counselling by Pregnancy Heart Team is required	Yes: expert counselling by Pregnancy Heart Team is required	Yes: expert counselling by Pregnancy Heart Team is required, with clear and thorough discussion of very high pregnancy risk and shared decision-making process for termination if pregnancy occurs
Obstetric and cardiac care during pregnancy	Local hospital	Local hospital	Shared care with local hospital + Pregnancy Heart Team	Care led by Pregnancy Heart Team	Care led by Pregnancy Heart Team
Location of delivery	Local hospital	Local hospital	Shared care with local hospital + Pregnancy Heart Team. Location depends on CV status and evolution of pregnancy	Expert centre, care led by Pregnancy Heart Team	Expert centre, care led by Pregnancy Heart Team

AHT, arterial hypertension; ARVC, arrhythmogenic right ventricular cardiomyopathy; ASI, aortic size index; BAV, bicuspid aortic valve; BrS, Brugada syndrome; CARPREG II, Cardiac Disease in Pregnancy study II; CPVT, catecholaminergic polymorphic ventricular tachycardia; CV, cardiovascular; DCM, dilated cardiomyopathy; EF, ejection fraction; HCM, hypertrophic cardiomyopathy; HTAD, heritable thoracic aortic diseases; INR, international normalized ratio; LQTS, long QT syndrome; LQT2, long QT syndrome type 2; LV, left ventricle; mWHO, modified World Health Organization; NDLVC, non-dilated left ventricular cardiomyopathy; NSTE ACS, non-ST-elevation acute coronary syndrome; NYHA, New York Heart Association; PAH, pulmonary arterial hypertension; PPCM, peripartum cardiomyopathy; RV, right ventricle; SCAD, spontaneous coronary artery dissection; STEMI, ST-elevation myocardial infarction; TR, tricuspid regurgitation.

[a]Definition of cardiac events: cardiac arrest, cardiac death, arrhythmia requiring treatment, left/right heart failure, thromboembolic event, aortic dissection, acute coronary syndrome, or hospitalization for cardiac reason. Endocarditis only in van Hagen *et al.*[51]



[b]Estimation of maternal adverse cardiac event rate with integration of CARPREG II score. Reprinted from Silversides *et al.*[52] with permission from Elsevier.

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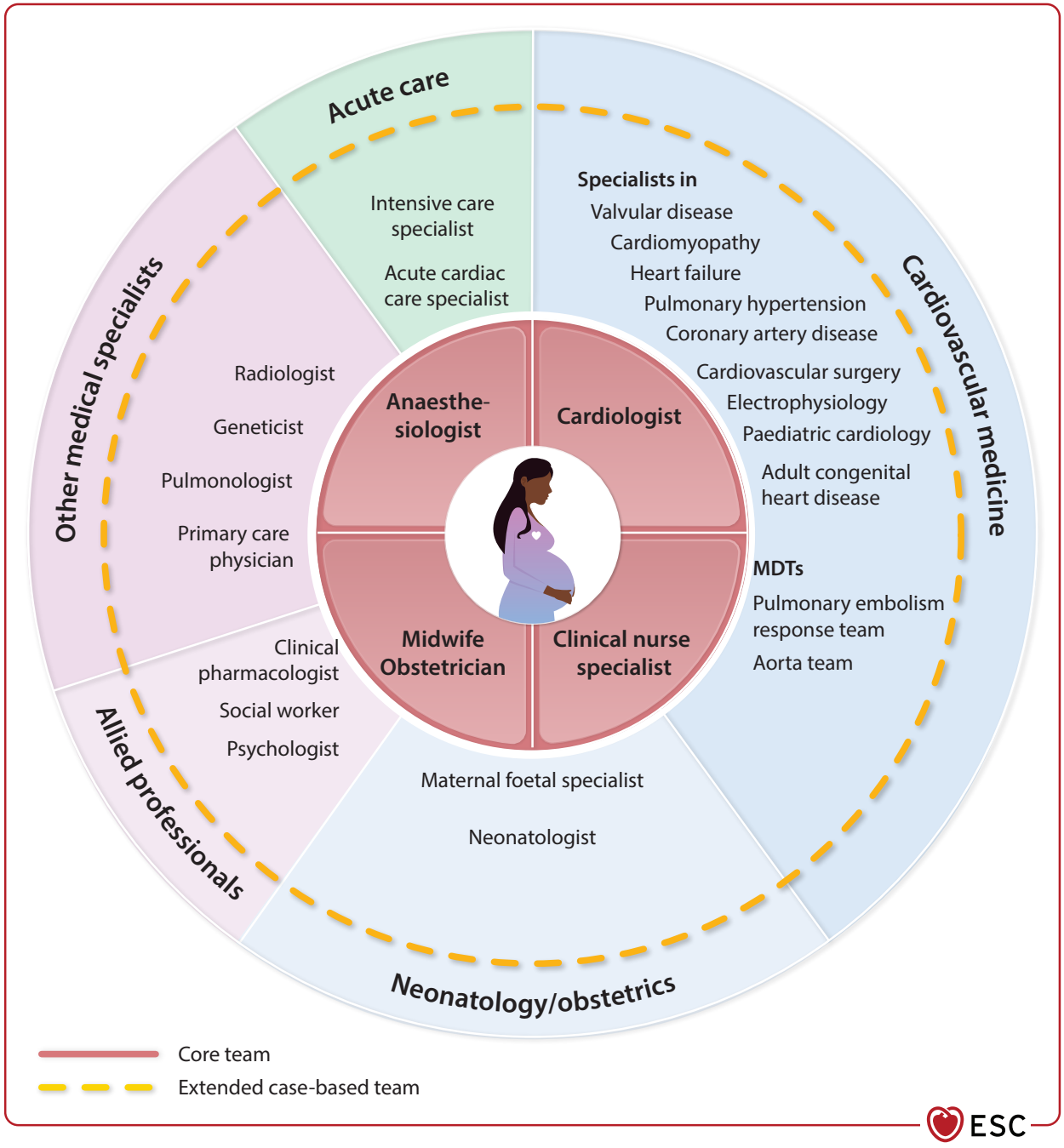

Figure 3 Composition of the core and expanded case-based Pregnancy Heart Team. MDT, multidisciplinary team.

Nevertheless, women with CVD requesting reproductive treatment should not be turned down based on assumed cardiovascular risk until their case has been discussed in a multidisciplinary setting involving the Pregnancy Heart Team.

Assisted reproduction has added risks above those of pregnancy alone; superovulation is pro-thrombotic and can be complicated by ovarian hyperstimulation syndrome, with marked fluid shifts and a high thrombosis risk. All women with CVD who are embarking on fertility treatment should have an individual risk assessment for venous thromboembolism (VTE) given the risk associated with these techniques.[69] The risk of ovarian hyperstimulation syndrome can be reduced by careful cycle monitoring, using a low-dose follicle-stimulating hormone in combination with a gonadotropin-releasing hormone antagonist. Transferring a single embryo is strongly advised in women with CVD, as carrying multiple gestations is associated with greater cardiovascular changes and more maternal and foetal complications.[70,71]

In women with mWHO 2.0 class III conditions or those who are anticoagulated (*Table 6*), the risk of complications from superovulation is very high. It is therefore recommended that these women have a full pre-pregnancy assessment by a Pregnancy Heart Team prior to the

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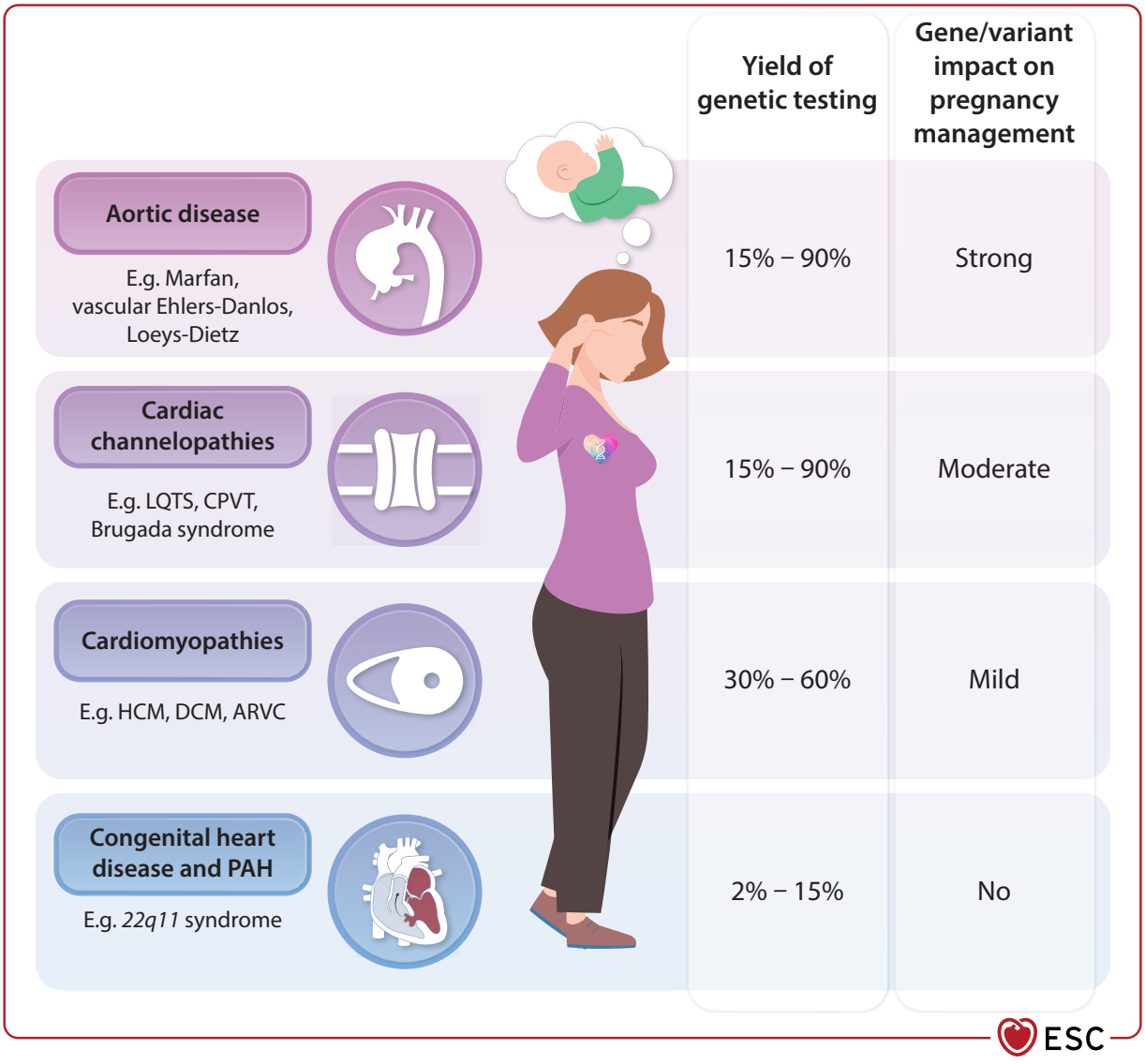

Figure 4 Pre-conception counselling and genetic aspects. ARVC, arrhythmogenic right ventricular cardiomyopathy; CPVT, catecholaminergic polymorphic ventricular tachycardia; DCM, dilated cardiomyopathy; HCM, hypertrophic cardiomyopathy; LQTS, long QT syndrome; PAH, pulmonary arterial hypertension.

Table 7 Pre-implantation and pre-natal options and implications

Pre-implantation genetic diagnosis	IVF procedure followed by biopsy and genetic testing of a single cell of the embryo. Embryo transfer with success rate of 25%–30% (dependent on mother's age and fertility).
	Risks to mother and offspring of IVF, such as multiple birth, premature labour and low birth weight, as well as side effects of hormonal treatment. Availability, expense and methods differ across countries.
Chorionic villus sampling	Transcervical or transabdominal sampling of the chorionic villi at the end of the first trimester.
	Procedure-related foetal loss rate ~0.2%.[66]
Amniocentesis	Direct sampling of amniotic fluid after 15 weeks of gestation.
	Procedure-related foetal loss rate ~0.1%.[66]

Adopted from the 2023 ESC Guidelines for the management of cardiomyopathies.[60]
IVF, *in vitro* fertilization.

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procedure, including an evaluation of the risk of hormonal treatment. The option of natural cycle IVF should be considered. Hysteroscopy and laparoscopy can be life-threatening procedures in women with specific cardiac conditions, such as a Fontan circulation, and should only be undertaken in an experienced centre with appropriate support.

Fertility treatment should be avoided in women with mWHO 2.0 class IV conditions.

4.2.4. Contraception

To ensure informed decision-making about pregnancy, accurate counselling about contraception should be provided to all girls and women of childbearing age with CVD, starting from menarche, to prevent unplanned pregnancies. An overview of the benefits and risks of different types of contraception in women with CVD is provided in *Table 8*.

4.2.5. Termination of pregnancy including psychological support

It is strongly recommended to consider and discuss termination of pregnancy with women whose risk is classified as mWHO 2.0 class IV due to the exceptionally elevated risk of maternal and foetal mortality or severe morbidity.[83] Efforts should be made to minimize delays for women seeking pregnancy termination because the risk of procedure-related complications increases as gestational age advances. Surgical methods are often preferred, but pharmacological methods remain an option until the ninth week of pregnancy.[83] Given the emotional and psychological impact of pregnancy termination, it is crucial to provide professional psychosocial support, which plays a significant role in reducing adverse mental health outcomes.[89] At the same time a discussion should be held regarding appropriate contraception.

Table 8 Overview of benefits and risks of different methods of contraception in women with cardiovascular disease

Method	Benefits	Cardiovascular risks	Cautious use and contraindications	Contraceptive efficacy
Hormonal oral contraceptives				
Progestin-only oral contraceptives	Minimal/no impact on coagulation factors Safe CV risk profile[72,73]	Mild fluid retention	LQTS not on beta-blockers[74,75]	++ *(general)* +++ *(for drospirenone)*[73]
Combined oral contraceptives[76,77]	Regular menstruation with reduced blood loss	VTE, hypertension and altered lipid profile[a]	Known dyslipidaemia[78] Pre-existing hypertension[79] Obesity[80] Cyanosis MHV Fontan circulation Risk factors for ACS[81]	+++
Long-acting reversible contraceptives				
Levonorgestrel-releasing IUD	↓ Menstrual bleeding and iron loss	None specified	Vasovagal responses on insertion and removal (done by gynaecologist) → *caution and monitoring with availability of anaesthesiologist recommended in PAH and Fontan circulation*[82]	Safest and most effective option +++
Smaller levonorgestrel IUD	↓ Menstrual bleeding and iron loss Easier to insert ↓ Risk of vasovagal responses	None specified	—	+++
Copper IUD	↓ Cost	—	↑ Intensity of menstrual bleeding	+++
Etonogestrel-releasing subcutaneous implants	No pelvic infection risk	None specified	Surgical subcutaneous insertion *(in the forearm with local anaesthesia—outpatient procedure)*	+++
Depot medroxyprogesterone acetate injection[83]	Lighter menses	Increased VTE risk, weight gain	Irregular bleeding	++
Barrier methods				
—	↓ Pelvic infection risk[84]	—	None specified	+

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Permanent sterilization				
Tubal ligation **Vasectomy**	Permanent	Anaesthetic and procedural risks	Non-reversible	+++
Emergency contraception				
Oral contraceptive pills to delay ovulation				
Ulipristal acetate	↑ Effectiveness than levonorgestrel	No ↑ thrombosis risk[85–87]	None specified	+++ *(only if taken before ovulation)*
Levonorgestrel *single dose of 1.5 mg <72 h after unprotected intercourse*	—	No ↑ thrombosis risk	None specified	++ *(only if taken before ovulation)*
Contraceptive device				
Copper IUD *<120 h after unprotected intercourse*	—	—	None specified	+++ *(in addition to ongoing contraception)*[85–88]

ACS, acute coronary syndrome; CV, cardiovascular; IUD, intrauterine device; LQTS, long QT syndrome; mg, milligram; MHV: mechanical heart valve; PAH, pulmonary arterial hypertension; VTE, venous thromboembolism.
↑ increase ↓ decrease.
[a]Higher in combined oral contraceptive pills containing ethyniloestradiol compared to natural oestradiol or oestetrol.

Recommendation Table 1 — Recommendations for counselling, pregnancy risk assessment, contraception, assisted reproductive technology, and the involvement of a Pregnancy Heart Team (see Evidence Table 1)

Recommendations	Class[a]	Level[b]
Maternal risk assessment		
It is recommended to perform a risk assessment in all women with CVD of childbearing age using the mWHO 2.0 classification[c].[44,45,47–49,54]	I	C
A discussion by the Pregnancy Heart Team about the high risk of maternal mortality or morbidity and the related high foetal risk is recommended for women with mWHO 2.0[c] class IV conditions, including a shared decision-making process for pregnancy termination, involving psychological support.[65]	I	C
It is recommended that women with CVD of mWHO 2.0[c] class II–III and above are evaluated and managed by a Pregnancy Heart Team from pre-pregnancy onwards through pregnancy and post-partum.[44,45,47–49,54]	I	C
Methods of contraception		
It is recommended that women with CVD of mWHO 2.0[c] class II and above, or those at risk of developing CVD, receive individualized advice to determine the most suitable contraception method, including emergency contraception.[90,91]	I	C
Progestin-only treatment, contraceptive implants, and/or levonorgestrel IUDs should be considered when there is any risk of thromboembolic events.[73,92–94]	IIa	B
Genetic counselling		
Assessment by a clinical geneticist prior to pregnancy is recommended in women fulfilling diagnostic criteria for inherited cardiovascular disease to guide risk stratification and pre-natal genetic testing.[63,95]	I	C
Pre-conception genetic counselling is recommended in couples with heritable CVD, whether genetic testing is being considered or not. It is recommended that this counselling is provided by an appropriately trained healthcare professional within a multidisciplinary team that offers psychological support and education to encourage decision-making.[63,95]	I	C
Reproductive technology		
It is recommended that single embryo transfer is performed in women with CVD.[70,71]	I	C
Pregnancy termination		
It is recommended to offer women with CVD access to termination of pregnancy that is tailored to their cardiac condition to minimize the risks of the procedure.[83]	I	C

CVD, cardiovascular disease; IUD, intrauterine device; mWHO, modified WHO.
[a]Class of recommendation.
[b]Level of evidence.
[c]The mWHO 2.0 classification is the updated mWHO classification from the 2018 ESC Guidelines for the management of cardiovascular diseases during pregnancy[43] and described in *Table 6*.

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4.3. Diagnostic methods in pregnancy

The pros and cons of the primary cardiovascular diagnostic methods for diagnosing cardiovascular disease in pregnancy are described here. Pre-pregnancy evaluation is covered in the risk assessment section (*Section 4.2.1*).

4.3.1. Electrocardiogram, including mobile rhythm devices

Pregnant women may present changes in their surface electrocardiogram (ECG), including increased heart rate, minor leftward QRS axis shift (15–20 degrees), slightly decreased (20 ms) PR interval, prominent Q waves in II, III, and aVF, and flat or inverted T-waves in III, aVF, V1, V2, and V3 (*Figure 1*).[96–98]

A 12-lead ECG is part of the standard evaluation of pregnant women presenting with new-onset cardiac signs or symptoms or suspected arrhythmia. In pregnant women presenting with syncope or palpitations, long-term Holter monitoring or implantable loop recorders should be considered as additional diagnostic tools. As pregnant women are more prone to arrhythmias, the threshold to perform long-term ambulatory rhythm monitoring should be low.

4.3.2. Echocardiography

Transthoracic echocardiography (TTE) is the first-line imaging method used in pregnancy.[99,100] Physiological changes in cardiac geometry and functioning are expected during pregnancy (*Figure 1*).[99,101–103] These are greatest early in the third trimester and resolve early post-partum. Agitated saline contrast should not be used during pregnancy, given the risk of placental infarction due to microbubble embolism, resulting in foetal distress.[99,104] Relevant foetal exposure to intravenous (i.v.) echocardiographic contrast agents is not expected due to their very short half-life.[105] Nevertheless, these agents should only be used selectively because studies during pregnancy or lactation are lacking.[99] Transoesophageal echocardiography is relatively safe, but the potential risks and benefits must be weighed individually, including the risk of emesis/aspiration and sudden increase in intra-abdominal pressure. Speckle-tracking echocardiography is a useful method to detect subclinical myocardial abnormalities in pregnancy.[106,107]

4.3.3. Cardiopulmonary exercise testing

If there is suspicion of new-onset CVD during pregnancy, submaximal exercise testing (at 80% of predicted maximal heart rate) can be useful to assess cardiovascular response to exercise. There is no evidence that exercise testing increases the risk of spontaneous miscarriage.[108,109] Stress echocardiography using bicycle ergometry may improve diagnostic specificity. The use of pharmacologic stress agents (e.g. dobutamine) should be avoided.[99,110] There is no evidence supporting a preference for treadmill over bicycle exercise testing during pregnancy. The choice should be based on women's individual risk factors, contraindications, pregnancy stage, and local availability of testing and expertise.

4.3.4. Biomarkers

Throughout pregnancy and the early post-partum stages, natriuretic peptide [NP: B-type natriuretic peptide (BNP) and N-terminal pro-brain natriuretic peptide (NT-proBNP)] values within the normal range have a strong negative predictive value for heart failure whereas the positive predictive value tends to be lower.[111,112] In women with pre-existing cardiomyopathy, ACHD, or valvular heart disease, baseline as a minimum and serial NP measurements on an individualized basis should be considered to diagnose cardiac complications during pregnancy and post-partum.[113–118]

Although cardiac troponin I (cTnI) and cardiac troponin T (cTnT) are essential in diagnosing myocardial ischaemia, standardized values in pregnancy and post-partum have not been established.[112,119] Therefore, routine use of troponins alone during pregnancy is not recommended.

D-dimer testing has relevance in the diagnosis of VTE (see *Section 11*), taking into account the physiological increase during pregnancy, particularly in the third trimester.

4.3.5. Ionizing radiation exposure

Risks of ionizing radiation exposure are highest during organogenesis and decrease with time.[120,121] Exposing the foetus to radiation doses >150–200 mGy may result in intrauterine growth restriction (IUGR), congenital malformations (in particular of the central nervous system), and malignancies. If possible, procedures should be delayed at least until the completion of major organogenesis (>12 weeks of gestation). However, the safety of the woman is important and should guide the clinical decision. All radiation doses to the foetus must be kept 'as low as reasonably achievable' (ALARA) (preferably <50 mGy) and should be clearly documented. Manoeuvres to minimize radiation are: (i) use echo guidance when possible; (ii) place the source as far and the receiver as close as possible to the patient; (iii) use only low-dose fluoroscopy (7.5 frames per second or lower); (iv) favour anteroposterior projections; (v) avoid direct radiation of the abdominal region (abdominal shielding is of limited benefit due to internal scatter from thoracic tissues rather than direct foetal irradiation); (vi) collimate as tightly as possible to the area of interest; (vii) minimize fluoroscopy time; and (viii) ensure the procedure is performed by an experienced cardiologist.[122–126] Iodinated contrast can cross the placenta, but has not been reported to have teratogenic effects.[127] The potential risk of congenital hypothyroidism is unclear but no abnormalities of foetal thyroid function after application have been reported.[122,128,129]

4.3.5.1. Chest radiography

The chest radiograph is a practical and readily available diagnostic tool for evaluating cardiopulmonary diseases. The foetal dose from chest radiography is <0.01 mGy. Nevertheless, it should only be performed in symptomatic women if other methods fail to clarify the cause of the symptoms.

Lung ultrasound is a valuable tool for diagnosing pleural effusion, pulmonary oedema, pneumothorax, and pneumonia. However, there is currently a lack of data about the regular ultrasound pattern during pregnancy,[130,131] and lung ultrasound is therefore not recommended as an alternative to chest radiography.

Protection of the foetus is governed by radiological standards. Both the technician and the radiologist should act accordingly.

4.3.5.2. Computed tomography and nuclear medicine imaging

The radiation dose to the foetus from a chest computed tomography (CT) or pulmonary CT angiography is estimated at 0.02 mGy.[121] Technetium-99m, used for ventilation–perfusion lung scanning for detection of pulmonary embolism, results in an embryonic or foetal exposure of <5 mGy, which is considered a safe dose in pregnancy. Computed tomography or nuclear medicine techniques are generally

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not recommended during pregnancy. However, if such techniques are necessary because other diagnostic tools are insufficient or not readily available for the diagnosis in question, they should not be withheld from a pregnant patient.[100,132]

4.3.5.3. Cardiac catheterization

Cardiac catheterization is seldom needed during pregnancy but may be necessary for specific diagnostic and interventional purposes. Foetal compromise decreases with gestational age. The highest risk is <20 weeks gestation and is proportional to the radiation dose, with no reports of foetal anomalies or loss when exposure is <50 mGy.[133,134] Most coronary procedures can be performed within these dose limits and radiation exposure to the foetus itself is estimated to be lower than 20%. The radial approach by an experienced operator is preferable and every effort to reduce radiation exposure should be made.

4.3.6. Cardiovascular magnetic resonance

Cardiovascular magnetic resonance (CMR) is advised if other non-invasive diagnostic measures are insufficient to provide a clinical diagnosis and is preferable to radiation-based imaging modalities.[99,100,135] It seems prudent to avoid a scanner strength higher than 1.5 tesla due to the greater energy deposition in tissue. Evidence regarding gadolinium-based contrast in pregnancy is controversial and its use should be avoided unless absolutely necessary.[135–137] Excretion of gadolinium-based agents into breast milk is limited (<0.04% of an i.v. dose within the first 24 h, with 1%–2% absorption).[138] Lactating women receiving intravascular gadolinium should discontinue lactation for 24 h.[133,138,139]

4.4. Foetal assessment

4.4.1. Risk of foetal/obstetric complications

The typical increase in CO during pregnancy may not occur optimally in some women with CVD, potentially affecting uteroplacental blood flow. These and other cardiovascular risk factors contribute to an increased risk of obstetric and foetal complications, including foetal loss, stillbirth, pre-term birth, pre-eclampsia, and IUGR.[140] Furthermore, the severity of obstetric and foetal outcomes varies depending on the maternal risk as defined in the mWHO 2.0 classification. Adverse outcomes are more frequent in women with a higher mWHO 2.0 classification, emphasizing the importance of risk stratification, comprehensive counselling, and multidisciplinary management, including neonatology expertise.[2,134,140] Notably, pulmonary hypertension (PH) represents one of the highest risks for obstetric and foetal complications.[141] The main predictors of neonatal complications are indicated in *Table 9*.

4.4.2. Screening for congenital heart disease in the foetus

Foetal echocardiography should routinely be offered at 18–22 weeks when parents have congenital heart disease. This will detect up to 80% of significant congenital cardiac defects.[142–144]

4.4.3. Assessing foetal well-being

Detailed anatomical foetal assessment is required in women using cardiac medication with teratogenic effects (see *Section 5* for more details).[145]

Recommendation Table 2 — Recommendations for diagnostic methods in pregnancy (see Evidence Table 2)

Recommendations	Class[a]	Level[b]
Echocardiography		
Transthoracic echocardiography is recommended as first-line imaging tool in any pregnant woman with unexplained or new cardiovascular signs or symptoms.[99]	I	C
Biomarkers		
Measurement of BNP and NT-proBNP levels should be considered prior to pregnancy in women with HF of any aetiology, including previous PPCM, cardiomyopathy, ACHD, and PAH, and be monitored during pregnancy according to the underlying disorder and in case of new-onset or worsening symptoms.[114]	IIa	B
Ionizing radiation		
It is recommended to limit exposure to all medical ionizing radiation doses to ALARA levels.[121]	I	C
It is recommended to keep the radiation dose to the foetus as low as possible (preferably <50 mGy), particularly if the foetus is in the field of view.[120,121]	I	C
A CT scan should be considered for PE when clinical benefits outweigh the risks to the mother and foetus.[100,121,132]	IIa	C
A chest radiograph may be considered as a first-line imaging tool if other methods are not successful in clarifying the cause of dyspnoea.	IIb	C
Coronary angiography with minimal radiation may be considered during pregnancy if potential benefits outweigh the risks.	IIb	C
Cardiovascular magnetic resonance		
Discontinuation of lactation for 24 h should be considered in women in whom i.v. gadolinium is required.[133,139]	IIa	C
CMR imaging without gadolinium contrast should be considered for a definitive, clinically relevant diagnosis during pregnancy, if other non-invasive diagnostic measures are not sufficient.[135,136]	IIa	C

ACHD, adult congenital heart disease; ALARA, as low as reasonably achievable; BNP, B-type natriuretic peptide; CMR, cardiovascular magnetic resonance; CT, computed tomography; HF, heart failure; i.v., intravenous; NT-proBNP, N-terminal pro-brain natriuretic peptide; PAH, pulmonary arterial hypertension; PE, pulmonary embolism; PPCM, peripartum cardiomyopathy.
[a]Class of recommendation.
[b]Level of evidence.

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Table 9 Predictors of neonatal events in pregnancies of women with cardiovascular disease

Predictors of neonatal events
NYHA class III/IV or cyanosis during baseline pre-natal visit
Maternal left heart obstruction
Low maternal oxygen saturation (<90%)
Multiple gestations
Use of anticoagulants
Cardiac medication before pregnancy
Mechanical valve prosthesis
Maternal cardiac event during pregnancy
Maternal decline in CO during pregnancy

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Derived from the 2018 ESC Guidelines for the management of cardiovascular diseases during pregnancy.[43]
CO, cardiac output; NYHA, New York Heart Association.

In women with beta-blocker exposure, higher small for gestational age (SGA) rates and, more rarely, bradycardia have been reported, indicating the need for appropriate foetal monitoring.[12,146] Foetal ductus venosus Doppler velocity is a useful adjunct to evaluate foetal well-being and determine the time to delivery in cases of increased risk of IUGR.[147]

4.4.4. Foetal assessment of heritable primary arrhythmias

In families with primary arrhythmia, the foetus may present with arrhythmias. Therefore, the foetal heart rate should be assessed at baseline and during each pre-natal visit and compared against gestation-specific norms. In pregnancies complicated by suspected primary arrhythmia-related foetal arrhythmias, complete foetal echocardiography, typically performed at 20–22 weeks, is recommended to evaluate heart anatomy, ventricular function, and the arrhythmia mechanism.[148] Foetal magnetocardiography, if available, offers valuable insights into arrhythmia type and severity and monitors anti-arrhythmic drug therapy, as it captures all cardiac time intervals (P, QRS, T-wave) between 17 and 24 weeks of gestation. It is currently the only method to detect repolarization abnormalities, such as QT interval prolongation.[149]

4.5. Timing and mode of delivery

An individualized delivery plan should be made that covers the needs for induction of labour, labour management, delivery, and post-partum surveillance, in shared decision-making with the pregnant women. This delivery plan should be widely accessible to the patient, her partner, and relevant health professionals, and should be placed in the patient's (electronic) health record.

4.5.1. Timing of delivery

Pregnant women with CVD are more likely to have comorbidities and experience adverse events during delivery than those without CVD, and require additional monitoring and care.[150] Any maternal benefit of early term delivery (from 37 weeks 0 days to 38 weeks 6 days of gestation) should be weighed against the increased likelihood of adverse foetal outcomes.[151] Induction of labour between 39 and 40 weeks reduces the risk of emergency caesarean section by 12% and the risk of stillbirth by 50% in women without CVD. The benefit is likely to be greater for women with CVD who have higher rates of obstetric complications.[152,153] In the absence of maternal or foetal indications for early birth, induction of labour before 39 weeks should be reserved for obstetrical indications.[154]

4.5.2. Induction of labour

Mechanical methods, prostaglandin E1 analogue (misoprostol), slow-release formulation of 10 mg prostaglandin E2 (dinoprostone), oxytocin, and artificial rupture of membranes are all considered safe to induce labour.[4,155,156] High-dose (600 mg) misoprostol does not affect cardiac parameters in women without heart disease, although there remains a theoretical risk of coronary vasospasm and arrhythmias.[155] Dinoprostone may cause profound hypotension, but only when injected blindly into the myometrium, and this route of administration should be avoided.[157] The use of an additional 2 IU of oxytocin for the management of the third stage in women with CVD has no cardiac consequences and is associated with significantly lower blood loss.[158] In women at high risk (mWHO 2.0 classes III–IV), oxytocin is generally considered as a first-line uterotonic, misoprostol and carboprost are second line (see Supplementary data online, *Table S3*).[159]

Mechanical methods such as a cervical ripening balloon might be preferable in women where a drop in systemic vascular resistance would be detrimental.[160] If membranes are ruptured, augmentation of labour should be immediate to reduce the risk of infection and should be undertaken with oxytocin to minimize the number of vaginal examinations.[4]

4.5.3. Vaginal or caesarean delivery

Vaginal delivery is associated with less blood loss and lower risk of infections and venous thromboembolism and should be advised for most women.[161] Planned caesarean section does not confer any advantage over planned vaginal delivery in terms of maternal outcomes and may be associated with adverse foetal outcomes.[162,163]

Caesarean section is the preferred mode of delivery for obstetric indications and for women presenting in labour who use or have used vitamin K antagonist (VKA) within the past 2 weeks, with high-risk aortopathy (mWHO 2.0 class III), with hypertrophic cardiomyopathy (HCM) and severe left ventricle outflow tract obstruction, or in acute intractable HF.[43]

4.5.4. Haemodynamic monitoring during delivery

Pulse oximetry, blood pressure monitoring, and continuous ECG monitoring may help detect early signs of decompensation, arrhythmias, and ischaemia in women with significant CVD and identify those in whom delivery should be expedited.[164] Arterial lines should be reserved for those women who have haemodynamic instability or are at risk of it. A right-heart catheter is of uncertain benefit, is associated with complications, and should be avoided in most cases. Minimally invasive CO monitoring is preferable, where possible.[110]

4.5.5. Anaesthesia/analgesia

Analgesia is crucial for labour in pregnant women with CVD to reduce physical stress. Neuraxial methods are very effective analgesic blocks. The onset of conventional epidural analgesia is relatively slow (±15 min) and allows for careful titration of a local anaesthetic–opioid mix.[165] Spinal analgesia is suitable for women with high-risk CVD, where a faster onset of sympathetic block is desirable.[166] Combined spinal–epidural analgesia typically has a faster onset time (±5 min). However, adverse effects such as hypotension and foetal heart rate abnormalities occur more quickly and are more pronounced. Different techniques for administering low-concentration, high-volume local anaesthetic–opioid regimens allow maintenance of epidural analgesia through the epidural

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catheter. Whenever an epidural catheter is *in situ* in a high-risk woman, higher doses can be administered for conversion to caesarean section, avoiding airway and other complications of general anaesthesia. In women at risk of dural ectasia, including Marfan syndrome, extra caution and management in an expert centre is essential. Furthermore, a pre-delivery consultation with the anaesthesia team is needed.[167] When neuraxial analgesia is contraindicated due to conditions such as systemic anticoagulation or spinal deformities, opioids (i.v. remifentanil) are an alternative despite the risk of hypoventilation and apnoea.[168,169] Single-shot spinal analgesia is common in caesarean delivery for its simplicity and effectiveness.[167]

4.5.6. Delivery in women on anticoagulants

4.5.6.1. Planned delivery

In women with mechanical heart valves (MHVs) taking VKAs, suspension of VKAs and bridging with heparin [either therapeutic-dose low-molecular-weight heparin (LMWH) or i.v. unfractionated heparin (UFH)] is recommended at least 2 weeks before planned delivery (see also *Section 5* and *Section 12*). This is because of the slow metabolism of VKA in the foetus. If therapeutic-dose LMWH is used, one strategy is to switch to i.v. therapeutic UFH at least 36 h before planned delivery.[170] In these settings the target activated partial thromboplastin time (aPTT) is ≥2 times control values. UFH can then be stopped 4–6 h before surgery (in case of caesarean section) or before insertion of regional anaesthesia or anticipated vaginal delivery. For women who are on therapeutic-dose LMWH for non-MHV indications, dosing can be omitted for 24 h prior to caesarean section or anticipated vaginal delivery with no need for bridging. In women with MHVs who are on LMWH and aspirin in combination, consideration should be given to stopping aspirin 4 days before delivery.[170]

4.5.6.2. Urgent delivery on therapeutic anticoagulation

Managing women who are anticoagulated during delivery is complex and needs an individualized approach. *Figure 5* gives an overview, but again each scenario may need a more tailored solution.

4.5.6.2.1. Delivery on vitamin K antagonists. If women require urgent delivery and have been taking VKAs within the last 2 weeks, then delivery by caesarean section is recommended to reduce the risk of foetal intracranial bleeding. When urgent delivery is required, preventing bleeding complications with administration of i.v. four-factor prothrombin complex concentrate (4F-PCC), depending on the international normalized ratio (INR) (25 U/kg for a therapeutic INR range of 2–4) is the preferred method for rapid INR normalization. If necessary, vitamin K should be given.[171,172] If 4F-PCC is not available, fresh frozen plasma (FFP) is an alternative, but it takes longer to reverse an elevated INR and requires a larger fluid challenge.[171,173]. The involvement of an expert haematologist in these scenarios is essential, in addition to the Pregnancy Heart Team. The foetus may remain anticoagulated for 8–10 days after discontinuation of maternal VKAs, and may need to be given FFP and higher doses of vitamin K.[170]

4.5.6.2.2. Delivery on heparin. If delivery occurs after recent administration of heparin (e.g. within 4–6 h of UFH, with non-normalized aPTT, or within 12 h of therapeutic LMWH) protamine sulfate should be given. Neutralization of LMWH varies between products and may be less effective.[174] Protamine dosage depends on timing after the last dose of LMWH (1 mg/1 mg enoxaparin <8 h; 0.5 mg/1 mg enoxaparin >8 h). For UFH, 1 mg of protamine per 100 units of heparin is needed.[175]

In addition to the level of anticoagulation, the decision to reverse anticoagulation should also be related to the bleeding risk, which is higher with conditions such as placental abruption, placenta previa, and multiple previous caesarean sections.

4.5.6.3. Restarting anticoagulation after delivery

The decision to restart anticoagulation post-delivery is challenging and must balance risk of bleeding and risk of thrombosis. Anaesthetic, cardiac, haematology, and obstetric teams may have different priorities, but all need to be actively involved in decision-making, which should also involve the patient. Late obstetric bleeding (>24 h) is common,[176] as was also confirmed in recent data from the ROPAC III trial[177] (bleeding on mean post-partum day 3.6), suggesting that these events occur at a time when heparin is being used at the same time as the VKA is being reintroduced. Restarting UFH (aPTT levels ≥2 times the control) or low/intermediate doses of LMWH are all valid options.[170] Techniques to reduce bleeding risk include active management of the third stage of labour with oxytocin. Recently, the effect of adding 2 IU oxytocin over 10 min to a standard treatment of low-dose infusion for 4 h [10 IU of oxytocin in 500 mL of normal saline given i.v. at 36 mL/h for 4 h (12 mU/min)] was analysed. The addition of 2 IU of oxytocin was not associated with any greater derangement in cardiovascular measures, but with a significantly lower volume of blood loss.[158] VKA should only be started 7–14 days or later post-partum to reduce the risk of late bleeding.[170]

4.5.7. Endocarditis prophylaxis for delivery

Systemic antibiotics according to the 2023 ESC Guidelines for the management of endocarditis may be considered for delivery in women at high risk of endocarditis.[178]

Recommendation Table 3 — Recommendations for timing and mode of delivery (see Evidence Table 3)

Recommendations	Class[a]	Level[b]
Timing and mode of delivery		
Vaginal delivery is recommended in most women with CVD.[161–163]	I	B
Systemic antibiotic prophylaxis may be considered for delivery in women at high risk[c].[178]	IIb	C
Routine induction of labour prior to 39 weeks is not recommended in women with stable CVD.[44,154]	III	C
Delivery in women on anticoagulants		
It is recommended that the timing of delivery is planned to ensure safe and effective peripartum anticoagulation.	I	C
It is recommended to discontinue VKAs and start therapeutic-dose LMWH or adjusted-dose i.v. UFH at the 36th week of gestation or 2 weeks before the planned delivery.[179]	I	C
In women at low risk[d] on therapeutic-dose LMWH, neuraxial anaesthesia and vaginal delivery (or caesarean section for obstetric indications) is recommended 24 h after the last dose of LMWH.[180]	I	C

Continued

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In women at high risk[d], it is recommended to convert LMWH to i.v. UFH at least 36 h prior to delivery and stop the UFH infusion 4–6 h prior to anticipated delivery. The aPTT should be normal before regional anaesthesia.[180]	I	C
If delivery starts while the mother is on VKAs or <2 weeks after discontinuation of VKAs, caesarean section is recommended for foetal protection.	I	C
Post-delivery, it is recommended that the decision to restart LMWH or UFH is made after discussion with the Pregnancy Heart Team and the woman who gave birth.[170]	I	C
It is recommended to postpone the switch from heparin back to oral anticoagulants until 7–14 days post-partum when the wound area has healed, in consultation with the Pregnancy Heart Team.[177]	I	C
In women on therapeutic-dose LMWH, planned delivery should be considered at around 39 weeks to avoid the risk of spontaneous labour while fully anticoagulated.	IIa	C
In women who are on antenatal anticoagulation, active management of the third stage of labour with oxytocin should be considered.[158]	IIa	C

aPTT, activated partial thromboplastin time; CVD, cardiovascular disease; i.v., intravenous; LMWH, low-molecular-weight heparin; UFH, unfractionated heparin; VKA, vitamin K antagonist.
[a]Class of recommendation.
[b]Level of evidence.
[c]Patients with prosthetic cardiac valves or a history of infective endocarditis, or cardiac transplant patients with residual valve defects.
[d]See *Table 10*.

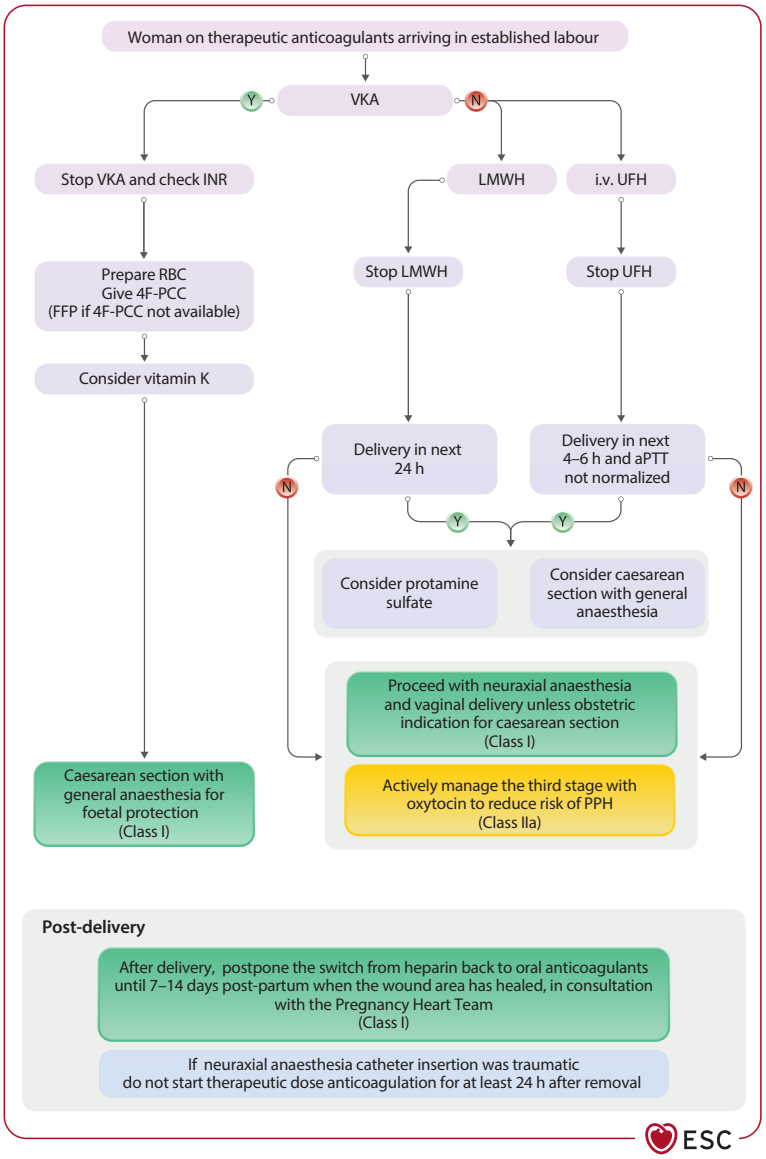
Woman on therapeutic anticoagulants arriving in established labour
VKA
Y
N
Stop VKA and check INR
LMWH
i.v. UFH
Prepare RBC
Give 4F-PCC
(FFP if 4F-PCC not available)
Stop LMWH
Stop UFH
Consider vitamin K
Delivery in next 24 h
Delivery in next 4–6 h and aPTT not normalized
N
Y
Y
N
Consider protamine sulfate
Consider caesarean section with general anaesthesia
Proceed with neuraxial anaesthesia and vaginal delivery unless obstetric indication for caesarean section (Class I)
Actively manage the third stage with oxytocin to reduce risk of PPH (Class IIa)
Caesarean section with general anaesthesia for foetal protection (Class I)
Post-delivery
After delivery, postpone the switch from heparin back to oral anticoagulants until 7–14 days post-partum when the wound area has healed, in consultation with the Pregnancy Heart Team (Class I)
If neuraxial anaesthesia catheter insertion was traumatic do not start therapeutic dose anticoagulation for at least 24 h after removal
ESC

Figure 5 Management of urgent delivery in women under anticoagulants. aPTT, activated partial thromboplastin time; FFP, fresh frozen plasma; INR, international normalized ratio; i.v., intravenous; LMWH, low-molecular-weight heparin; 4F-PCC, four-factor prothrombin complex concentrate; N, no; PPH, post-partum haemorrhage; RBC, red blood cell; UFH, unfractionated heparin; VKA, vitamin K antagonist; Y, yes.

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4.6. Post-partum monitoring and complications

4.6.1. Monitoring

The post-partum period is associated with significant haemodynamic changes and fluid shifts. Hence, women are at risk of adverse outcomes, such as hypertension, HF, or stroke.[181,182] Post-partum management must be individualized and depends on the woman's underlying CVD, risk or presence of arrhythmias and HF symptoms, and the course during pregnancy and delivery. For women at the highest HF risk or with HF symptoms during pregnancy or delivery, admission to an intensive (cardiac) care unit during the first 24–48 h for haemodynamic monitoring should be considered.[183] Early ambulation is important to reduce the thromboembolism risk.

In women with hypertensive disorders of pregnancy, blood pressure should be monitored in hospital (or with an equivalent level of outpatient surveillance) for 72 h after birth and checked again 7–10 days post-partum. Optimizing blood pressure levels from the immediate post-partum period until the first post-natal months could help prevent the development of hypertension and improve long-term cardiovascular health.[184]

4.6.2. Breastfeeding and lactation

Throughout these Guidelines, the term 'lactation' (including not only breastfeeding but also other methods such as pumping) is used as the default term in most sections, especially where it encompasses a broader scope, such as in medication-related contexts. We use 'breastfeeding' specifically in sections where the focus is on the act of nursing or direct feeding at the breast, particularly when discussing its physiological and long-term health outcomes.

Breastfeeding is a global priority because interruption of lactation is associated with adverse health outcomes for the woman and her child, including higher maternal risks of breast cancer, ovarian cancer, diabetes, and hypertension, and greater infant risks of infectious and metabolic disease.[185,186]

Inhibition of lactation can be obtained with standard doses of cabergoline in general, or bromocriptine in peripartum cardiomyopathy (PPCM).

Several drugs are contraindicated during lactation (see *Figure 6* in *Section 5* and Supplementary data online, *Table S4*).

4.6.3. Complications

4.6.3.1. Haemorrhage

Post-partum haemorrhage (PPH) is more frequently reported in women with CVD.[187] To reduce the risk of PPH, an active third stage of labour with early cord clamping and administration of oxytocin to deliver the placenta should be pursued. Maternal anaemia is a known risk factor for PPH, so anaemia should be managed aggressively in the antenatal period.

At the time of delivery, a slow i.v. infusion of 2 IU oxytocin over 10 min immediately after birth, followed by 12 mU/min for 4 h, reduces the PPH risk and has a minimal impact on cardiovascular parameters.[158] In cases of PPH that are refractory to medical treatment, additional devices may be used, such as the Bakri intrauterine balloon, uterine compression sutures, or further haemostatic measures including uterine artery embolization or hysterectomy.

4.6.3.2. Psychological reactions, post-partum depression

Although the general risk of post-partum depression among new mothers in the general population is ~10%–20%, this risk increases with underlying health conditions such as CVD, where ~1 in 3 mothers have reported symptoms of depression in the post-partum period.[188] Those with PPCM are particularly vulnerable to depression.[189–191] These findings emphasize the critical need for early detection, regular mental health screening and the necessity of holistic care models with psychological support and tailored interventions.[191,192]

5. Drugs during pregnancy and lactation

5.1. General principles

5.1.1. Pharmacokinetics and pharmacodynamics in pregnancy

Physiological adaptation of maternal organ systems to pregnancy affects the pharmacokinetics and pharmacodynamics of potentially all medical treatments, including cardiovascular drugs (see *Section 3.2*).[146,193,194]

5.1.2. Pharmacogenetics

An overlap between individual genotypes associated with drug effects and pregnancy-induced modifications (e.g. liver enzymes) may unmask adverse effects or require careful titration, in particular for drugs that lead to severe adverse drug reactions (ADRs). As an example, warfarin can cause severe ADR at the maternal (bleeding, thrombosis) and/or foetal level (embryotoxicity, teratogenesis).[195] The most notable cases refer to the polymorphisms of *CYP2D6*, associated with different phenotypes (extensive, ultrarapid, or poor metabolizers), leading to diverse pharmacokinetics/pharmacodynamics of drugs used in pregnancy such as beta-blockers (e.g. labetalol, metoprolol), antidepressants (e.g. fluoxetine, paroxetine), and analgesic drugs (e.g. tramadol, codeine).[194] Poor and ultrarapid metabolizers may experience extreme variations in drug plasma level and bioavailability, and hence in their effects.[195]

5.1.3. Newborn drug exposure in breast milk

The exposure of the newborn to maternal drugs via breast milk is expressed as a percentage value, calculated as the dose taken by the infant compared either to the therapeutic dose of the same drug (often unknown for newborns) or the maternal weight-adjusted dose.[196] The 'relative infant dose' (RID) depends on the relative amount of drug secreted in the milk (milk-to-plasma concentration ratio) and the quantity of milk intake (the standard is 150 mL/kg/day) on a body weight basis.[196] The dose per kg of the infant is compared to the maternal dose per kg over the same period. A RID lower than 5%–10% is generally considered safe (see *Figure 6*; Supplementary data online; *Table S4*; and LactMed database).[197]

5.2. Drug classes in pregnancy

5.2.1. Anticoagulants

The use of anticoagulants during pregnancy represents a complex balance of risks and benefits, influenced by specific indications, and hampered by low-quality evidence. Indications for anticoagulation in pregnancy are diverse and covered in different sections in these Guidelines. In this section, we cover drug-specific aspects and dosing regimens that explicitly pertain to the setting of pregnancy.

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Table 10 List of anticoagulation regimens and disease entities in which they are indicated

Indication	Type of anticoagulant	Dosing	Timing
Low thrombosis risk			
VTE prevention/no indication for oral anticoagulation[a]	LMWH	Prophylactic dose	o.d.
Uncomplicated Fontan circulation[b]	LMWH	Prophylactic dose	o.d.
Intermediate thrombosis risk			
VTE (DVT/PE) during pregnancy[a]	LMWH	Therapeutic dose	o.d. or b.i.d.
Persistent/permanent AF at elevated thromboembolic risk[c]	LMWH	Therapeutic dose	o.d. or b.i.d.
Decreased ventricular function (EF <35%) and/ or intracardiac thrombus[d]	LMWH	Therapeutic dose	o.d. or b.i.d.
High thrombosis risk			
Mechanical heart valves[e]			
1. First trimester			
Low VKA dose to achieve required INR[f]	First trimester: VKA or LMWH	INR: weekly to every 2 weeks	
		LMWH: dose adjusted to peak anti-factor Xa level	b.i.d.
High VKA dose to achieve required INR	Switch to LMWH	Dose adjusted to peak anti-factor Xa level (weekly until threshold, every 2–4 weeks thereafter)	b.i.d.
2. From week 13: shared decision			
(a) Continue/switch to VKA with weekly to every 2 weeks INR			
(b) Continue LMWH with dose adjustment as above			
Delivery: refer to *Section 4.5.6.2.* (for urgent delivery) and *Section 4.5.6.1* (for planned delivery)			

AF, atrial fibrillation; b.i.d., is in die (twice a day); DVT, deep vein thrombosis; EF, ejection fraction; INR, international normalized ratio; LMWH, low-molecular-weight heparin; o.d., omni die (once a day); PE, pulmonary embolism; VKA, vitamin K antagonist; VTE, venous thromboembolism.
[a]Refer to *Section 11* Venous thromboembolism.
[b]Refer to *Section 9* Pregnancy in women with congenital heart disease.
[c]Refer to section 12.4.1.2. Atrial fibrillation including anticoagulation.
[d]Refer to section 12.6 Heart failure.
[e]Refer to *Section 12.5.3.2* Mechanical heart valves.
[f]≤5 mg of warfarin; ≤2 mg/day acenocoumarol, ≤3 mg/day phenprocoumon.[43]

Low-molecular-weight heparin treatment regimen glossary

To ensure consistency throughout these Guidelines, we apply the following wording about LMWH treatment—any deviations from these standards are clearly indicated in the specific sections:

- *Prophylactic-dose LMWH* refers to low fixed doses with adjustment for extremes of body weight.[198]
- *Therapeutic-dose LMWH* refers to high doses typically reserved for treatment of VTE (*Section 11*) and thrombosis prevention in MHVs (*Section 12*).

An individualized shared decision-making approach with careful consideration of maternal thrombosis risk vs foetopathy is needed, and strategies will vary from prophylactic dosing of LMWH to correctly dosed VKAs. Regional differences, also related to lower availability of anti-factor Xa level monitoring in low- and middle-income countries as indicated by the ROPAC III study, also need to be taken into account. Interestingly, this study shows that despite higher monitoring and better resource availability, the risk of thrombosis was higher in high-income countries, specifically due to higher use of treatment regimens with therapeutic LMWH.[177] Haemorrhagic complications in the mother can occur with all regimens.[199] *Table 10* lists the regimens and disease entities in which anticoagulants are indicated.

5.2.1.1. Vitamin K antagonists

Vitamin K antagonists cross the placenta and are associated with embryopathy and foetopathy risk, even at low doses. They will therefore be switched to LMWHs in most pregnant women, with the only exception being women with atrial fibrillation (AF) in the context of moderate to severe mitral valve stenosis or MHVs, given the lower thrombosis risk with VKAs compared to LMWH in the latter (see *Section 12*).[179,200] Women receiving chronic VKAs who are contemplating pregnancy need counselling regarding avoidance of the potential teratogenic effects. When switching (usually to LMWH) is desired, this should take place as soon after conception as possible.

Vitamin K antagonist embryopathy is thought to be related to interference with embryonic ossification.[201,202] Adverse impact is highest in the first trimester (0.6%–12% of embryopathy) and much lower but persisting in later stages of pregnancy (0.7%–2% risk of foetopathy, e.g. central nervous anomalies, intracranial haemorrhage).[200,203,204] The risk of embryopathy in the first trimester depends on the VKA dose. The risk was 0.45%–0.9% in pregnancies with low-dose warfarin according to two systematic reviews.[199,205] In this setting, low-dose refers to the dose necessary to maintain the appropriate INR (according to current guidelines this equals doses of ≤5 mg of warfarin, ≤2 mg/day acenocoumarol, ≤3 mg/day phenprocoumon).[43] This approach may be seen as a reasonable balance between the risks to the mother with

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Table 11 Dosing regimens for the commonly used low-molecular-weight heparins

	Enoxaparin	Dalteparin	Tinzaparin	Target
Prophylactic LMWH *Body weight 50–100 kg*	4000 IU o.d.	5000 IU o.d.	4500 IU o.d.	NA
Therapeutic LMWH (non-MHV)	150 IU/kg o.d.	200 IU/kg o.d.	175 IU/kg o.d.	NA
Therapeutic LMWH MHV	125 IU/kg b.i.d. (starting dose) then 100 IU/kg b.i.d.	125 IU/kg (starting dose) b.i.d. then 100 IU/kg b.i.d.	250 IU /kg (starting dose) then 175 IU/kg o.d.	0.8–1.2 U/mL anti-factor Xa (4–6 h post administration)

b.i.d., bis in die (twice a day); IU, international units; LMWH, low-molecular-weight heparin; MHV, mechanical heart valve; NA, not applicable; o.d., omni die (once a day).

MHV and the foetus.[170,200,204,206,207] Due to incomplete development of liver metabolism, the INR takes longer to normalize in the foetus and neonate than in the mother, which is why VKA should be discontinued 2 weeks before delivery (see *Section 4*).

If the indication of anticoagulation is non-MHV, such as pregnancy-related VTE, VKAs are not recommended. In case of pre-existing VKA or direct oral anticoagulant (DOAC) therapy due to previous VTE, VKAs and DOACs should be replaced by LMWH when pregnancy is planned or at recognition of pregnancy.[208]

Vitamin K antagonists are safe during lactation[209] and are recommended in all women with MHVs given their superior anticoagulant properties in avoiding valve thrombosis.[179,200]

5.2.1.2. Low-molecular-weight heparins

Embryopathy or foetopathy has not been reported with LMWHs, even in therapeutic doses, but thromboembolic complications in women with MHVs are higher than with VKAs (8.7%, 5.8%, and 2.7% for LMWH, UFH, and VKA, respectively).[179,200] LMWHs appear less likely to induce heparin-induced thrombocytopenia compared with UFH, although this has not been studied in pregnancy.[210]

Data on optimal dosing and frequency of administration in pregnancy are scarce and mostly limited to the setting of VTE and MHV.

5.2.1.2.1. Low-molecular-weight heparin dosing. In women with MHVs, slightly higher starting doses are suggested to ensure minimal delay in reaching the target range (See *Table 11*).[176]

For prophylactic-dose LMWH, a fixed low-dose LMWH regimen can be used in most cases.[207] In women with acute VTE requiring therapeutic LMWH dose, routine anti-factor Xa monitoring has not been shown to affect clinical outcomes despite fluctuations of anti-factor Xa levels during pregnancy, and should only be considered in women with renal insufficiency or obesity, where adjustment for body weight may result in overdosing.[211–213] Underweight patients show a low prevalence of antepartum or post-partum VTE[214] and do not require specific recommendations compared to patients with normal weight.[215]

Monitoring of anti-factor Xa levels is essential in women with MHVs on therapeutic-dose LMWH: at least weekly until target level is achieved or when there is a below target level at any stage, and regular monitoring thereafter (e.g. every 2–4 weeks depending on stability) (see *Section 12.5.3.2*). Recommended peak anti-factor Xa levels should be individualized based on type and location of the valve (between 1.0 and 1.2 U/mL) and additional trough level measurement may be indicated in selected cases with increased thrombosis risk (see *Section 12.5.3.2*).[216]

5.2.1.2.2. Once-daily vs twice-daily administration. In pregnant women with confirmed acute VTE, no clear benefit of a twice-daily LMWH administration vs a once-daily administration has been demonstrated.[217,218] Thus, either using a once- or twice-daily regimen, each one resulting in a therapeutic dose, is reasonable.

Twice-daily administration at slightly higher doses is the usual therapeutic dosing regimen for pregnant women with MHVs.[177] There is insufficient evidence for the use of LMWH injections more frequently than twice daily.

5.2.1.3. Unfractionated heparin

Intravenous UFH, although not crossing the placenta, is associated with higher risks of thrombocytopenia and osteoporosis compared with LMWH. The risk of valve thrombosis during pregnancy with subcutaneous UFH is unacceptably high and its use is not recommended.[219]

In women with MHVs in whom VKAs cannot be continued, intravenous UFH is only indicated when anti-factor Xa monitoring is not possible during the first trimester and at the time of delivery (see *Section 4.5.6*). However, intravenous heparin dosing is challenging, requiring hospitalization and multiple daily blood tests to achieve an aPTT ≥2 times control values.

5.2.1.4. Fondaparinux

In women requiring prophylaxis of VTE, good outcomes with subcutaneous fondaparinux were reported in an observational study of 65 pregnancies and a retrospective analysis in 84 women with one or more previous pregnancies.[220,221] Its use can be considered if there is an allergy or adverse response to LMWH (prophylactic dose: 2.5 mg daily; therapeutic dose: up to 10 mg daily)[221–223] (see *Section 11*).

5.2.1.5. Direct oral anticoagulants

Direct oral anticoagulants have shown better bleeding profiles than a LMWH or VKA regimen across diverse indications in non-pregnant populations. Outcome data on their use in pregnancy are scarce and inconsistently captured in pharmacovigilance databases, indicating a need for a more robust system of reporting.[224,225] The foetal effects of DOACs are controversial.[226,227] Animal and *in vitro* studies showed that dabigatran, rivaroxaban, and apixaban crossed the placenta.[228–230] Prescription information based on these data reported variable adverse effects in pregnant rodents and rabbits: post-implantation loss, maternal bleeding, or malformation at >4 times the recommended maternal doses (see Supplementary data online, *Table S4*). Counselling women on DOACs who are planning a pregnancy is advised, considering the complexity of pre- and post-conceptional switches to alternative

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regimens (LMWH, VKA) and the risk of VTE recurrence.[227] DOACs may have an edge over VKAs, such as rapid reversal in case of premature delivery and a short antepartum interruption period, due to their reversible inhibition of procoagulant factors. The oral route is an advantage over LMWHs. However, evidence of safety is lacking for specific DOAC antidotes (andexanet alfa, idarucizumab) in pregnant women and can only be inferred from pre-clinical studies. After uncertain initial reports on foetotoxicity,[224] a recent retrospective cohort study (mainly in women exposed to rivaroxaban) does not support a high risk of embryotoxicity.[231] It should be highlighted that despite promising studies, clinical evidence on the benefits and risks of DOACs for the mother and foetus is scarce and needed, and their foetal safety over VKAs during the second and third trimesters has not been established. DOACs are not recommended in pregnancy and they should only be used in the absence of any other option in consultation with the Pregnancy Heart Team and the haematology team. Based on current data there is no absolute indication to interrupt pregnancy in the case of accidental exposure.[227,232]

During lactation, alternative drugs should be preferred to DOACs due to the paucity of data. However, there are relevant differences between the agents. In studies on lactating women treated with apixaban, the concentration in milk was significantly higher than that of rivaroxaban: the milk-to-plasma ratio was >12%,[233] and the weight-adjusted infant doses 14%–20%.[234] Dabigatran etexilate mesylate, the orally available prodrug, is poorly excreted to the milk after biotransformation to dabigatran, and its oral absorption by the neonatal gastrointestinal tract is likely negligible. In two breastfed neonates of women receiving dabigatran, the maximum drug concentrations in the neonates' plasma were 100 000 times below the levels that would have a significant effect on coagulation indices.[235] In lactating women treated with 15–20 mg/day rivaroxaban, the breastfed infant would receive a low dose, corresponding to 1.3%–5% of the maternal weight-adjusted dosage.[233,236–238] Therefore, dabigatran and rivaroxaban may be taken cautiously during lactation. Signs of bleeding should be monitored in neonates of lactating mothers taking dabigatran.

Recommendation Table 4 — Recommendation for direct oral anticoagulants and pregnancy

Recommendation	Class[a]	Level[b]
DOACs are not recommended during pregnancy.	III	C

DOAC, direct oral anticoagulants.
[a]Class of recommendation.
[b]Level of evidence.

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5.2.2. Antiplatelet treatment

No teratogenic effect is reported for aspirin doses up to 300 mg daily. Clopidogrel is considered safe if dual antiplatelet therapy (DAPT) is needed for the shortest possible duration.[239,240] Ticagrelor is contraindicated due to embryotoxicity. Prasugrel may be considered during pregnancy in special populations including poor metabolizers in whom the prodrug clopidogrel has limited effect.[241,242] The use of glycoprotein IIb/IIIa inhibitors (eptifibatide and tirofiban) should only be used in pregnancy if strictly necessary.[240]

5.2.3. Diuretics and SGLT2 inhibitors

Diuretics may be used in pregnancy to treat systemic hypertension especially in emergencies or HF-related volume overload conditions. Care must be taken to monitor for reduction in plasma volume or CO, and decrease in placental perfusion.

Pre-clinical data on SGLT2 inhibitors showed that they cross the placenta[243] and exposure to these drugs may cause foetal damage in rodents, especially during the second and third trimesters.[244] SGLT2 inhibitors should be stopped before pregnancy and during lactation.

5.2.4. Pulmonary hypertension

Parenteral prostaglandin analogues (i.v. epoprostenol, treprostinil) can be used in pregnant women with significant right ventricle (RV) dysfunction, while recognizing that these agents may interfere with platelet aggregation and may promote bleeding.[245] Oral phosphodiesterase 5 inhibitors (e.g. sildenafil, tadalafil) can also be used, while recognizing the potential side effects of pre-term delivery and SGA babies.[246] Combination therapy with sildenafil and inhaled iloprost has also been reported.[247] Calcium channel blockers should be continued during pregnancy for women with vasodilator-responsive PAH and normal RV function. Endothelin receptor antagonists (ERAs, e.g. bosentan, ambrisentan, macitentan) should not be used in pregnancy due to their teratogenic potential. There are few data on the safety of agents such as bosentan and sildenafil in the post-partum period for lactating mothers; however, successful cases have been reported.[248,249]

5.2.5. Anti-arrhythmic agents

For women without structural heart disease, anti-arrhythmic drugs (such as flecainide, sotalol, and ibutilide) can be used for the prevention or termination of AF and atrial flutter (AFL).[250,251] Beta-blockers are considered safe, especially lipophilic compounds (labetalol, metoprolol, and propranolol). In pregnant women with AF and concomitant congestive HF, digoxin may be an alternative option for rate control. Amiodarone can cause foetal abnormalities, bradycardia, and thyroid dysfunction, and its routine use is contraindicated during pregnancy, but may be used as single dose in emergencies like ventricular tachycardia (VT) storm. There are no restrictions on amiodarone use in cardiac arrest.[252]

5.2.6. Calcium channel blockers

The safety and efficacy of nifedipine [the originator of dihydropyridine calcium channel blockers (CCBs)] as an antihypertensive in pregnancy has largely been proved in comparison with other antihypertensive treatments (see *Section 12.3*). A meta-analysis of 22 randomized control trials with 2595 participants found that nifedipine was significantly more effective at reducing patients' high blood pressure compared with other antihypertensive drugs (labetalol, hydralazine, methyldopa) in hypertensive patients.[253] Foetal, neonatal, and maternal safety outcomes were not statistically different between nifedipine and comparators, except for maternal headache and flushing.[253] A randomized controlled trial compared oral regimens with nifedipine, labetalol, or methyldopa in women requiring antihypertensive therapy due to severe hypertension. It found that the primary outcome of blood pressure control within 6 h with no adverse outcome was more common with nifedipine or labetalol than with methyldopa[254]. Amlodipine showed safety and efficacy similar to nifedipine.[255] Studies on the non-dihydropyridine CCB diltiazem are inadequate and significant potential teratogenic effects have been demonstrated in rodents and rabbits. The drug passes in milk, reaching relevant infant concentrations. Therefore, diltiazem is not recommended in pregnancy and lactation. Oral verapamil is considered safe; no teratogenicity has been observed. The drug is excreted at low levels in milk, <1% of the mother's weight-adjusted dosage.

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5.2.7. Renin–angiotensin–aldosterone system inhibitors

Angiotensin-converting enzyme inhibitors (ACE-Is), angiotensin receptor blockers (ARBs), angiotensin receptor/neprilysin inhibitors (ARNIs), and renin inhibitors can cause foetal malformations, IUGR and death, and are contraindicated in pregnancy. Caution should be recommended to childbearing women, especially in the absence of effective contraception. Captopril, enalapril, and benazepril are safe during lactation,[146] whereas ARBs are not recommended. Candesartan may be an exception.[256] Aldosterone antagonists, canrenone, and spironolactone can have anti-androgenetic effects and are contraindicated in pregnancy. Spironolactone is considered safe during lactation because of extensive metabolism to canrenone, thus the infant would receive less than 1% of the mother's daily dosage of canrenone.[257] Case reports of eplerenone in pregnant women with resistant hypertension identified no adverse effects.[258–261]

5.2.8. Lipid-lowering agents

Diagnosis of maternal hypercholesterolaemia at the first trimester or familial hypercholesterolaemia have adverse consequences for both foetus and mother.[262] Low-density lipoprotein (LDL) levels increase by ~30%–50%, high-density lipoprotein cholesterol by 20%–40%, and triglycerides by 50%–100% during pregnancy[262], so referring to reference range as for routine testing is of limited clinical use. Previously, lipid-lowering treatment was usually discontinued during pregnancy because of limited safety data.[43] Having been contraindicated in pregnancy since 1987, statins now remain contraindicated only during lactation. In July 2021 the United States Food and Drug Administration (FDA)[263] stated that the evidence was insufficient to conclude that a risk of miscarriage is increased with statins and requested removal of the contraindication.[264] Continuing with statins may therefore be considered during pregnancy in women with familial hypercholesterolaemia or established atherosclerotic cardiovascular disease (ASCVD) (see *Section 12.2*).[265] Furthermore, inadvertent conception during statin therapy does not require pregnancy termination but should prompt close follow-up. Bile acid binding sequestrants[265] and LDL apheresis[265] can be considered in women with familial hypercholesterolaemia. PCSK9 inhibitors and ezetimibe are not recommended during pregnancy due to lack of clinical data.[266] Bempedoic acid has a strong contraindication and therefore women are recommended contraception during its use.

5.2.9. Beta-adrenergic blocking agents

Beta-blocker use during early pregnancy has not been associated with an increased risk of congenital malformations.[250,267,268] Recent data from ROPAC indicate higher SGA rates in women with beta-blocker exposure (15.3% vs 9.3%, $P < .001$). With metoprolol as reference, labetalol (0.2, 95% CI 0.1–0.4) was the least likely to cause SGA, and atenolol (2.3, 95% CI 1.1–4.9) the most.[12] Labetalol and lipophilic drugs (metoprolol, propranolol, carvedilol) are preferred due to high first-pass metabolism, as well as beta-1-selective drugs (bisoprolol, metoprolol), which reduce the risk of hypoglycaemia in addition to reduced IUGR. Nadolol and pindolol are also safe in the case of arrhythmic events in cardiomyopathies and channelopathies (see *Section 6*).[269–271] Of note, the metabolism of metoprolol (and perhaps other oral lipophilic beta-blockers) was significantly higher in mid and late pregnancy than post-partum, likely due to enzymatic induction during pregnancy.[272] Changes in dosage (dose and frequency) are likely required if inadequate clinical responses are encountered.[272]

Atenolol causes severe growth restriction, bradycardia, and hypoglycaemia and is not recommended.[250,268,272,273]

Propranolol, metoprolol (combined with hydralazine), and labetalol had the lowest and sotalol the highest risk of neonatal bradycardia during lactation.[274] For the lipophilic beta-blockers, milk level was <1% of the maternal weight-adjusted dose, thus reducing the risks associated with neonatal exposure during lactation.[272]

5.2.10. Immunosuppressants

The balance between maternal and foetal safety is challenging for immunosuppressant therapy, especially for women with heart transplantation. Medication can pass to the milk and expose neonates and infants to adverse drug effects. Changes in maternal physiology impact the pharmacokinetics of immunosuppressant drugs (see *Section 3.2*).[275] Calcineurin inhibitors (e.g. cyclosporine, tacrolimus), mammalian target of rapamycin inhibitors (e.g. everolimus, sirolimus), and azathioprine are the drugs of choice during pregnancy and lactation, which should not be discouraged. Mycophenolate derivatives increase the risk of miscarriage and foetal malformations especially during the first trimester and should be discontinued at least 6 weeks before conception.[276,277]

5.2.11. Neuroactive drugs

Selective serotonin reuptake inhibitors, including sertraline, can be taken safely during pregnancy and lactation.[278] Zuranolone, a synthetic form of the neurosteroid allopregnanolone, has recently been approved for the treatment of post-partum depression. No information is available on its safety in patients with CVD. Zuranolone is excreted in the milk and lactation should be avoided in the absence of safety data.[279]

5.2.12. Obstetric drugs in patients with cardiovascular disease

Drugs for inducing ovulation, including follicle-stimulating hormone and luteinizing hormone or combinations, are associated with an increased risk of deep vein thrombosis (DVT) and PE, due to the sharp rise in oestrogen levels during follicle recruitment,[280] but no other immediate cardiovascular side effects are known.

5.3. Internet databases

See Supplementary data online, Internet databases.

5.4. List of drugs

See Supplementary data online, *Table S4*.

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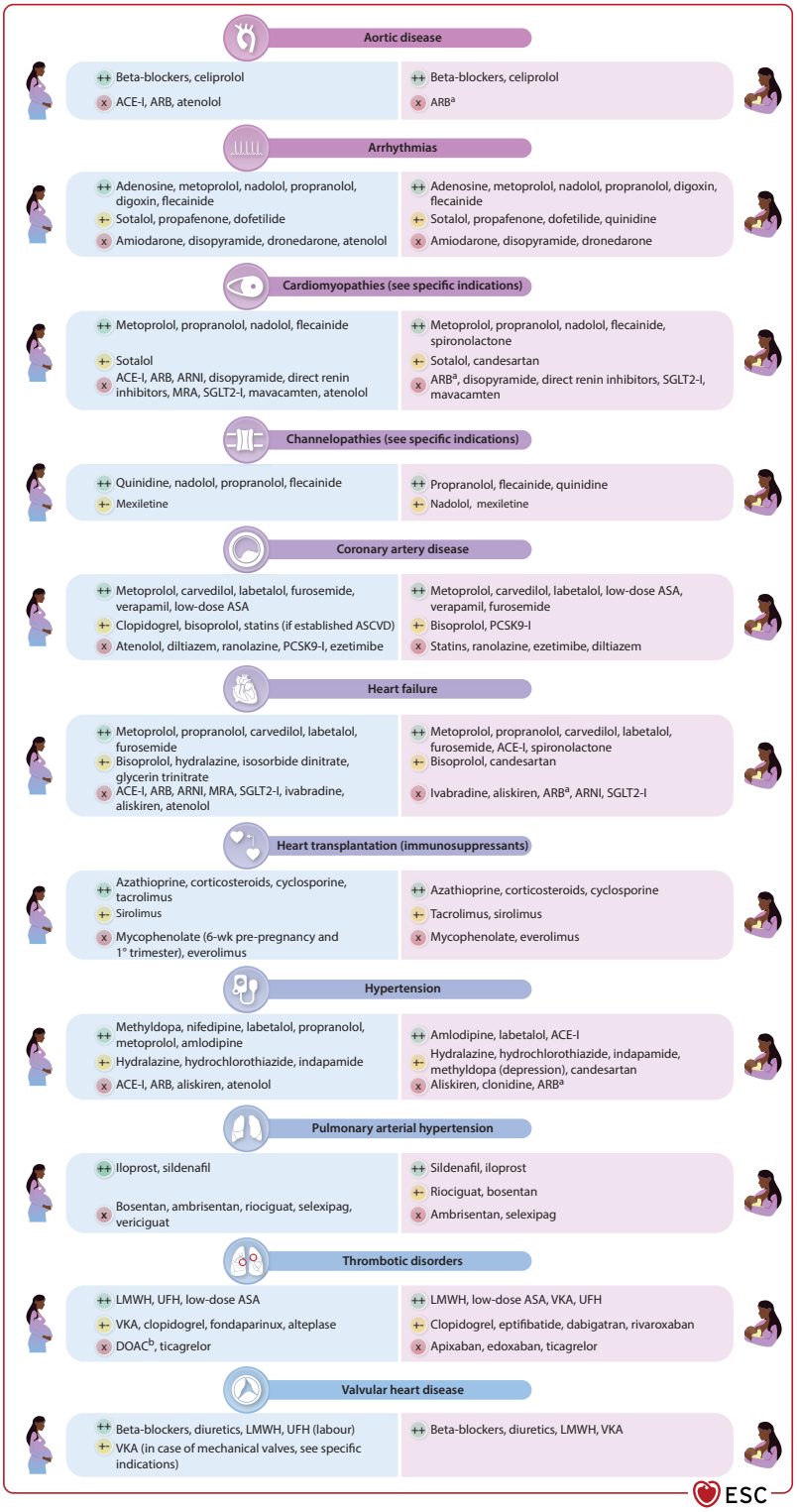
Aortic disease
++ Beta-blockers, celiprolol
x ACE-I, ARB, atenolol
++ Beta-blockers, celiprolol
x ARB[a]
Arrhythmias
++ Adenosine, metoprolol, nadolol, propranolol, digoxin, flecainide
+- Sotalol, propafenone, dofetilide
x Amiodarone, disopyramide, dronedarone, atenolol
++ Adenosine, metoprolol, nadolol, propranolol, digoxin, flecainide
+- Sotalol, propafenone, dofetilide, quinidine
x Amiodarone, disopyramide, dronedarone
Cardiomyopathies (see specific indications)
++ Metoprolol, propranolol, nadolol, flecainide
+- Sotalol
x ACE-I, ARB, ARNI, disopyramide, direct renin inhibitors, MRA, SGLT2-I, mavacamten, atenolol
++ Metoprolol, propranolol, nadolol, flecainide, spironolactone
+- Sotalol, candesartan
x ARB[a], disopyramide, direct renin inhibitors, SGLT2-I, mavacamten
Channelopathies (see specific indications)
++ Quinidine, nadolol, propranolol, flecainide
+- Mexiletine
++ Propranolol, flecainide, quinidine
+- Nadolol, mexiletine
Coronary artery disease
++ Metoprolol, carvedilol, labetalol, furosemide, verapamil, low-dose ASA
+- Clopidogrel, bisoprolol, statins (if established ASCVD)
x Atenolol, diltiazem, ranolazine, PCSK9-I, ezetimibe
++ Metoprolol, carvedilol, labetalol, low-dose ASA, verapamil, furosemide
+- Bisoprolol, PCSK9-I
x Statins, ranolazine, ezetimibe, diltiazem
Heart failure
++ Metoprolol, propranolol, carvedilol, labetalol, furosemide
+- Bisoprolol, hydralazine, isosorbide dinitrate, glycerin trinitrate
x ACE-I, ARB, ARNI, MRA, SGLT2-I, ivabradine, aliskiren, atenolol
++ Metoprolol, propranolol, carvedilol, labetalol, furosemide, ACE-I, spironolactone
+- Bisoprolol, candesartan
x Ivabradine, aliskiren, ARB[a], ARNI, SGLT2-I
Heart transplantation (immunosuppressants)
++ Azathioprine, corticosteroids, cyclosporine, tacrolimus
+- Sirolimus
x Mycophenolate (6-wk pre-pregnancy and 1° trimester), everolimus
++ Azathioprine, corticosteroids, cyclosporine
+- Tacrolimus, sirolimus
x Mycophenolate, everolimus
Hypertension
++ Methyldopa, nifedipine, labetalol, propranolol, metoprolol, amlodipine
+- Hydralazine, hydrochlorothiazide, indapamide
x ACE-I, ARB, aliskiren, atenolol
++ Amlodipine, labetalol, ACE-I
+- Hydralazine, hydrochlorothiazide, indapamide, methyldopa (depression), candesartan
x Aliskiren, clonidine, ARB[a]
Pulmonary arterial hypertension
++ Iloprost, sildenafil
x Bosentan, ambrisentan, riociguat, selexipag, vericiguat
++ Sildenafil, iloprost
+- Riociguat, bosentan
x Ambrisentan, selexipag
Thrombotic disorders
++ LMWH, UFH, low-dose ASA
+- VKA, clopidogrel, fondaparinux, alteplase
x DOAC[b], ticagrelor
++ LMWH, low-dose ASA, VKA, UFH
+- Clopidogrel, eptifibatide, dabigatran, rivaroxaban
x Apixaban, edoxaban, ticagrelor
Valvular heart disease
++ Beta-blockers, diuretics, LMWH, UFH (labour)
+- VKA (in case of mechanical valves, see specific indications)
++ Beta-blockers, diuretics, LMWH, VKA
ESC

Figure 6 Choice of medication during pregnancy (left) and during lactation and breastfeeding (right). ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor/neprilysin inhibitor; ASA, acetylsalicylic acid; ASCVD, atherosclerotic cardiovascular disease; DOAC, direct oral anticoagulant; LMWH, low-molecular-weight heparin; MRA, mineralocorticoid receptor antagonist; PCSK9-I, proprotein convertase subtilisin/kexin type 9 inhibitor; SGLT2-I, sodium–glucose co-transporter-2 inhibitors; UFH, unfractionated heparin; VKA, vitamin K antagonist; wk, week.

++ First/safest choice in pregnancy, lactation and breastfeeding. +- Second choice in pregnancy, lactation, and breastfeeding. x Evidence of foetal or infant toxicity or no data on safety. [a]Except candesartan. [b]See text for details.

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6. Pregnancy in women with cardiomyopathies and primary arrhythmia syndromes

6.1. Cardiomyopathies

Cardiomyopathies are characterized by disease-specific structural abnormalities and increased risk of ventricular and supraventricular arrhythmias. The risk associated with pregnancy in a woman with cardiomyopathy can be estimated using the mWHO 2.0 classification (*Table 6*). Pre-pregnancy, women with cardiomyopathies should be clinically evaluated to optimize treatment, avoid contraindicated drugs, and assess the risk of heart failure and arrhythmias. Indicated procedures, including implantable cardioverter defibrillator (ICD) implantation, should be performed before pregnancy. [60]

Genetic counselling is recommended before pregnancy to explain the probability of genetic transmission, risks for the mother, foetus, and child, and the possibilities of pre-implantation and pre-natal genetic testing (*Table 7*).[60,281] Women with cardiomyopathies should be managed by the Pregnancy Heart Team, including a cardiologist with expertise in cardiomyopathies and arrhythmias.

6.1.1. Dilated cardiomyopathy and non-dilated left ventricular cardiomyopathy

In women with dilated cardiomyopathy (DCM) and non-dilated left ventricle (LV) cardiomyopathy (NDLVC), severe systolic LV dysfunction, New York Heart Association (NYHA) functional class III/IV, RV failure, sustained ventricular arrhythmias, AF, and/or severe mitral valve regurgitation (MR) are high-risk criteria for major adverse cardiovascular events during pregnancy.[282] In contrast, women with mild LV dysfunction, good functional status, no arrhythmias, and no history of cardiac events are likely to have an uncomplicated pregnancy.[282]

Therapy should be modified before pregnancy. ACE-Is, ARBs, mineralocorticoid receptor antagonists (MRAs), sacubitril/valsartan, and SGLT2 inhibitors are all contraindicated during pregnancy. Pre-pregnancy risk stratification should include temporary withdrawal of contraindicated medication with close monitoring (see *Section 12.6* Heart failure). Beta-blockers should be continued with close monitoring of foetal growth. If anticoagulation is needed for AF or evidence of an intracardiac thrombus, LMWH should be used (see *Section 5* for dosing regimens).

Data about genotype-specific management during pregnancy are scarce but one study evaluated the risk of pregnancy and progression of cardiomyopathy in women with lamin A/C (*LMNA*) P/LP variants.[283] A small subset of women experienced arrhythmias during pregnancy although a history of pregnancy was not associated with long-term adverse disease progression.[283]

More information is included in *Sections 7, 12.4*, and *12.6*.

6.1.2. Arrhythmogenic right ventricular cardiomyopathy

Several observational studies and registries have shown that pregnancies in women with arrhythmogenic right ventricular cardiomyopathy (ARVC) are generally well tolerated with good foetal outcomes and no cardiac mortality when receiving optimal surveillance and therapy.[284,285] Delivery was usually vaginal and appeared safe. Sustained ventricular arrhythmias were reported in 5% of pregnancies and HF in 13%. Neither increased arrhythmia burden or ICD shocks were observed.[284] Beta-blocker therapy should be continued during pregnancy (with the exception of atenolol) or could be started in pregnancy if needed. The two most used anti-arrhythmic drugs in previous studies, beside beta-blockers, were flecainide and sotalol. Both drugs have a long record of safety. However, sotalol should be used with caution in women with reduced ejection fraction (EF) and with careful corrected QT interval (QTc) monitoring.[60] Sotalol also has a beta-blocker effect, necessitating monitoring of foetal growth.[286,287] Amiodarone is contraindicated in pregnancy. In women at high arrhythmic risk, an ICD should be implanted, preferably before pregnancy (see *Section 12.4*).

Two large studies of women with ARVC showed that pre-pregnancy phenotypical severity, rather than pregnancy itself, was the primary risk factor. Pregnancy was uneventful in the overwhelming majority.[288,289] Pregnancy did not seem to accelerate long-term progression of the ARVC phenotype.[288]

6.1.3. Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy[290] is the most common inherited cardiomyopathy and is often caused by variants in sarcomeric genes.[60] Phenocopies such as Anderson–Fabry disease and Danon disease[290] are X-linked and therefore the cardiac phenotype tends to be milder and occurs later in life in females than males, making pregnancy usually uneventful.[291–293]

Despite higher maternal mortality in women with HCM compared with the general population, absolute maternal mortality is low (0.5%) and confined to women at particularly high risk (*Table 6*).[294,295] Data from the ROPAC registry showed that despite overall good outcomes, 23% of pregnant women with HCM developed major cardiac events, including VT (10%) and AF (1.7%), mostly in women already identified as high risk prior to pregnancy.[296] A recent systematic review including 1624 women confirmed low neonatal mortality (0.2%) and stillbirths (1%) in pregnant women with HCM.[297] A study including 242 women with HCM found that pregnancy was not a modifier of the long-term outcomes and pregnancy was well tolerated.[298] Risk factors for major adverse cardiovascular events were advanced NYHA class and higher age at diagnosis.[298] Left atrium diameter as a risk factor has been reported with conflicting results.[298,299]

6.1.3.1. Treatment of hypertrophic cardiomyopathy in pregnancy

Ongoing beta-blocker therapy should be continued during pregnancy. Atenolol should be replaced before pregnancy (*Section 5.2.6*). Atrial fibrillation is poorly tolerated in HCM patients in general due to the risk of haemodynamic decompensation, and medical or electrical cardioversion of AF during pregnancy should be considered. Beta-blockers should be started during pregnancy when new symptoms occur [e.g. due to left ventricular outflow tract obstruction (LVOTO)], for rate control in AF, and to suppress ventricular arrhythmias. Verapamil is the second choice of drug when beta-blockers are not tolerated.

6.1.3.2. Left ventricular outflow tract obstruction

Left ventricular outflow tract (LVOT) gradients may increase slightly during pregnancy and were previously associated with increased cardiac events including arrhythmias and HF.[300] However, subsequent studies have not confirmed this association.[296,298]

In women with obstructive HCM, it is recommended to evaluate gradient in basal condition, with exercise and the Valsalva manoeuvre, before pregnancy and with only the medications allowed during pregnancy, to identify those needing septal reduction therapy before pregnancy.[60] Disopyramide may cause uterine contractions and is not recommended in pregnancy and should be discontinued unless the benefits outweigh foetal risk. Data on the safety of alcohol septal ablation during pregnancy are limited to a few case reports.[60,301,302]

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Myosin inhibitors (e.g. mavacamten) have not been tested in pregnancy and animal studies have shown foetal toxicity.[303] Therefore, contraception is recommended while on this treatment. Mavacamten may interact with hormonal contraception and therefore adding intrauterine or barrier contraception to hormonal contraception may be considered. Myosin inhibitor treatment should be discontinued at least 6 months before planning pregnancy[304] (see Supplementary data online, *Table S4*).

6.1.4. Hypertrabeculation of the left ventricle

Hypertrabeculation in isolation identified during pregnancy can be the simple consequence of an increased preload, can resolve after pregnancy, and cannot be used to make a diagnosis of cardiomyopathy.[60] Patients with hypertrabeculation and associated HCM, DCM, or NDLVC should follow the same recommendation as patients with the specific cardiomyopathy.[60]

6.1.5. Labour and delivery in cardiomyopathies

Labour and delivery may be associated with acute pain, adrenaline release, and need for urgent administration of anaesthetic drugs, and therefore haemodynamic monitoring and continuous telemetry monitoring during and after delivery is often warranted. In the absence of obstetric contraindications, vaginal delivery is generally recommended. Neuraxial anaesthesia reduces pain and therefore reduces adrenergic activation and arrhythmic risk.

In HCM, peripheral vasodilatation is poorly tolerated in women with severe LVOT obstruction (LVOTO) and therefore epidural and spinal anaesthesia should be applied cautiously. Low-risk LVOTO cases may have a spontaneous labour and vaginal delivery. Caesarean section may be the preferred option in women with severe LVOTO.

Recommendation Table 5 — Recommendations for cardiomyopathies and pregnancy

Recommendations	Class[a]	Level[b]
Clinical cardiological surveillance (ECG, echocardiogram, and Holter ECG monitoring) is recommended during pregnancy in women with CMPs, depending on individual risk.	I	C
Vaginal delivery is recommended in most women with CMPs, unless there are obstetric indications for caesarean section, severe HF (EF <30% and/or NYHA class III/IV), uncontrolled arrhythmias, or severe outflow obstruction (≥50 mmHg) in women with HCM, or in women presenting in labour on VKAs.[60]	I	C
Continuation of beta-blockers[c] should be considered during pregnancy in women with CMPs, with close follow-up of foetal growth.[60]	IIa	C
Dilated cardiomyopathy		
In women with DCM and worsening of EF during pregnancy, counselling on the risk of recurrence during a subsequent pregnancy is recommended in all cases, even after recovery of LV function.[43]	I	C
Arrhythmogenic right ventricular cardiomyopathy		
Flecainide, in addition to beta-blockers, should be considered as the anti-arrhythmic drug of choice in pregnant women with ARVC.[148]	IIa	C
Sotalol may be considered as an anti-arrhythmic drug in pregnant women with ARVC, with careful evaluation of QTc and while monitoring for foetal bradycardia and foetal growth and neonate hypoglycaemia.	IIb	C
Hypertrophic cardiomyopathy		
It is recommended to use the same risk stratification protocol for ventricular arrhythmias in pregnant women with HCM as for non-pregnant women with HCM.[43]	I	C
It is recommended to start beta-blockers[c] in women with HCM who develop symptoms due to outflow tract obstruction or arrhythmia during pregnancy.[43]	I	C
It is recommended that women with HCM with symptomatic LV dysfunction (EF <50%) and or severe LVOTO (≥50 mmHg) wishing to become pregnant are counselled by the Pregnancy Heart Team regarding the high risk of pregnancy-related adverse events.[294,295]	I	C
Cardioversion for AF should be considered in pregnant women with HCM.[43,305]	IIa	C
Disopyramide may be considered in pregnant women with HCM only when the potential benefits outweigh the risk of uterine contractions.[305,306]	IIb	C
Myosin inhibitors are not recommended in women during pregnancy due to lack of safety data.[303,304]	III	C

AF, atrial fibrillation; ARVC, arrhythmogenic right ventricular cardiomyopathy; CMP, cardiomyopathy; DCM, dilated cardiomyopathy; ECG, electrocardiogram; EF, ejection fraction; HCM, hypertrophic cardiomyopathy; HF, heart failure; LV, left ventricular; LVOTO, left ventricular outflow tract obstruction; NYHA, New York Heart Association; QTc, corrected QT interval; VKA, vitamin K antagonist.
[a]Class of recommendation.
[b]Level of evidence.
[c]Except for atenolol.

6.2. Primary arrhythmia syndromes

In general, women with primary arrhythmia syndromes tolerate pregnancy well. Genetic counselling is recommended before pregnancy, as discussed in *Section 6.1*. A complete clinical re-evaluation, optimization of treatment, and ICD evaluation should be undertaken prior to pregnancy.[252] Indicated treatment should be continued throughout pregnancy and in the post-partum period.

6.2.1. Long QT syndrome

Long QT syndrome (LQTS) is the most common channelopathy.[307] All patients with LQTS should take beta-blockers, with propranolol and nadolol being the most effective.[308] Additional therapies include left cardiac sympathetic denervation[309] and mexiletine for LQT3[252] and LQT2.[310,311] The foetal risk of mexiletine treatment is unknown, and decisions on treatment during pregnancy should be a shared decision with the woman.

Retrospective studies[312–316] show that women with LQTS were not at higher risk of cardiac events during pregnancy itself, but had an increased risk

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in the post-partum period (up to 12 months), especially for those with LQT2. Beta-blocker therapy was associated with risk reduction in all the studies. Women with LQTS should therefore start beta-blockers at pregnancy or continue beta-blockers at pre-pregnancy dose, with propranolol or nadolol as drugs of choice. Beta-blockers should be continued in the post-partum period, particularly in the case of women with LQT2 due to the increased arrhythmic risk. It should be noted that nadolol has a higher excretion in breast milk than propranolol, with a relative infant dose of 4%–7%.[317] Therefore, nadolol is generally not the preferred beta-blocker during lactation. However, arrhythmic risk can be high in LQTS, and nadolol is one of the drugs of choice; thereofore, a careful weighting of benefit against harm is appropriate. Change of beta-blocker therapy after delivery should be avoided, as this is a vulnerable phase. Therefore, a change from nadolol to propranolol should ideally be evaluated before pregnancy. High dosages of nadolol during lactation may require monitoring of the infant for bradycardia.

Women with LQTS should always avoid QT-prolonging drugs (see www.crediblemeds.org)[318]. Women with LQTS should be promptly treated for hypokalaemia and hypomagnesaemia, which is relevant in pregnancy-related hyperemesis, causing electrolyte disturbances and failure to absorb oral medications. All anti-emetic medications are QT-prolonging. Electrocardiogram monitoring should be performed if anti-emetic therapy is absolutely required.

Long QT syndrome can manifest very early in life, even during the foetal period, and can be a cause of stillbirth[319,320] (*Section 4.5.1*). A neonatal ECG should be performed post-delivery and after 2 weeks to avoid over-diagnosis due to transiently prolonged QT interval during the first 7–10 days of life.[321] Genetic screening for familiar genetic variants should be performed as soon as possible (e.g. from chordal blood). If the newborn is affected by LQTS, beta-blocker therapy should be started immediately.

6.2.2. Brugada syndrome

Men with Brugada syndrome (BrS) are more often symptomatic than women.[322] The only retrospective study on pregnant women with BrS did not show an increased risk of cardiac events during pregnancy and the post-partum period.[323] All patients with BrS should avoid contraindicated drugs (see www.brugadadrugs.org),[324] large meals, or excess alcohol, and promptly treat fever and its causes.[325] If there are symptoms during pregnancy, quinidine therapy should be considered with monitoring of hepatic function and blood count in the mother.

6.2.3. Catecholaminergic polymorphic ventricular tachycardia

Catecholaminergic polymorphic ventricular tachycardia (CPVT) is mainly caused by P/LP in the *RYR2* gene.[252] The only retrospective study published, involving 96 women and 228 pregnancies, did not show an increased risk of cardiac events during pregnancy and the post-partum period.[326]

Beta-blockers are the mainstay of therapy, with additive flecainide if needed.[322,327] Nadolol and propranolol are beta-blockers of choice and should be continued during pregnancy and lactation. As in LQTS, the higher excretion of nadolol in breast milk should be noted (*Section 6.2.1*). Left cardiac sympathetic denervation is a valuable anti-arrhythmic option that should be performed in experienced centres before pregnancy if indicated.[328] Implantable cardioverter defibrillators are indicated in a minority of patients with CPVT.

6.2.4. Short QT syndrome

Short QT syndrome (SQTS) is a rare channelopathy characterized by short QT and increased risk of life-threatening arrhythmias and AF.[329] There are no case reports or studies published on pregnancy in women with SQTS. When choosing an anti-arrhythmic drug during pregnancy, quinidine is the best option in the absence of more robust data.[330]

6.2.5. Labour and delivery in primary arrhythmia syndromes

In all primary arrhythmia syndromes, delivery should be planned with heart rhythm monitoring, electrolyte control, and post-operative ECG monitoring until all anaesthetic drugs have been eliminated.

Labour and delivery may be associated with acute pain, adrenaline release, and urgent administration of anaesthetic drugs, and therefore continuous telemetry monitoring is often warranted. In the absence of obstetric contraindications, vaginal delivery is generally recommended. Neuraxial anaesthesia reduces pain and therefore adrenergic activation which is specifically important in LQTS and CPVT. Women with LQTS and CPVT should continue beta-blocker therapy during labour and delivery. In women with CPVT, it is reasonable to keep the heart rate under the threshold for premature ventricular contraction onset during delivery, typically 100–110 b.p.m.[331,332] Anaesthetic drugs for LQTS should be selected according to the CredibleMeds website (www.crediblemeds.org).

In BrS, propofol and local anaesthetics with sodium-blocking agents (e.g. lidocaine) carry a theoretical risk of triggering arrhythmias. Case reports have indicated uneventful delivery with neuraxial anaesthesia in women with BrS.[333] Thiopental and inhalation anaesthesia have so far not been associated with adverse events.[324,333] Anaesthestic drugs should be chosen according the BrugadaDrugs website (www.brugadadrugs.org).

Recommendation Table 6 — Recommendations for primary arrhythmia syndromes and pregnancy (see Evidence Tables 4–6)

Recommendations	Class[a]	Level[b]
Monitoring and treatment of hypokalaemia and hypomagnesaemia is recommended in pregnant women with primary arrhythmia syndromes suffering from hyperemesis.[334]	I	C
Long QT syndrome		
Beta-blockers[c], with pre-pregnancy dose and with nadolol and propranolol as drugs of choice, are recommended during pregnancy in women with LQTS.[312–316,335]	I	B
It is recommended to continue beta-blocker therapy during lactation in women with LQTS to reduce arrhythmic risk.[148,336]	I	B
Pre-pregnancy beta-blocker dose of nadolol or propranolol, is recommended in women with LQT2, particularly in the post-partum period, which represents a high-risk period for life-threatening arrhythmias.[148,313,315]	I	B

Continued

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In women carrying a LQTS P/LP variant and who are phenotype-negative, use of beta-blockers[c] during pregnancy, post-partum, and lactation should be considered.[148]	IIa	C
Left cardiac sympathetic denervation should be considered before pregnancy in high-risk woman with LQTS who are not adequately protected by pharmacological therapies or who have appropriate ICD shocks despite optimal medical therapy.[252]	IIa	C
Brugada syndrome		
Quinidine therapy should be considered in pregnant women with BrS who have arrhythmic events during pregnancy.[337,338]	IIa	C
Catecholaminergic polymorphic ventricular tachycardia		
Beta-blockers[c], with pre-pregnancy dose and with nadolol and propranolol as drugs of choice, are recommended during pregnancy and lactation in women with CPVT.[43,148,252]	I	C
Flecainide, in addition to beta-blockers, is recommended in women with CPVT who experience cardiac events such as syncope, VT, or cardiac arrest during pregnancy.	I	C
It is recommended that women with CPVT who are stable on beta-blockers (nadolol or propranolol as drugs of choice) and flecainide before pregnancy continue both drugs during pregnancy and post-partum.	I	C
The use of beta-blockers[c] during pregnancy and lactation should be considered in phenotype-negative women with a CPVT P/LP variant.[148]	IIa	C
Left cardiac sympathetic denervation should be considered before pregnancy in high-risk women with CPVT who are not adequately protected by pharmacological therapies or with appropriate ICD shocks despite optimal medical therapy.[252]	IIa	C
Short QT syndrome		
It should be considered to continue quinidine therapy in women with SQTS throughout pregnancy and the post-partum period.[148]	IIa	C
Quinidine therapy should be considered in pregnant women with SQTS and arrhythmic events during pregnancy.[148]	IIa	C

BrS, Brugada syndrome; CPVT, catecholaminergic polymorphic ventricular tachycardia; ICD, implantable cardioverter defibrillator; LQTS, long QT syndrome, LQT2, long QT syndrome type 2; P/LP, pathogenic/likely pathogenic; SQTS, short QT syndrome; VT, ventricular tachycardia.
[a]Class of recommendation.
[b]Level of evidence.
[c]Except for atenolol.

7. Peripartum cardiomyopathy

7.1. Epidemiology

Peripartum cardiomyopathy is a potentially life-threatening condition defined as HF with reduced left ventricular ejection fraction (LVEF) <45%, without any other cause of HF, that occurs mainly during the peripartum period or in the months following delivery, termination, or miscarriage.[339] Peripartum cardiomyopathy is essentially a diagnosis of exclusion and requires urgent management.

Worldwide, PPCM is a complication of 1 of 2000 births,[340] but incidence rates vary depending on the geographical region, ethnicity, and socioeconomic factors, with an incidence of 1–4/1000 births in the United States of America[341,342] and 10/1000 births in the north-western region of Nigeria.[343] Recent data from 49 countries showed that most women presented with PPCM in the post-partum stage.[344–346] Risk factors for PPCM are shown in *Figure 7*.

7.2. Mechanisms

Recent trials suggest a 'multiple hit' theory for developing PPCM, with an accumulation of genetic and environmental risk factors (*Figure 7*). An overrepresentation of genetic variants in *TTN*, *FLNC*, *BAG3*, and *DSP* genes have been found in up to 15% of women with PPCM, with *TTN* truncating variants being the most common.[347,348] The prevalence of these four genes in women with PPCM was comparable to the prevalence in DCM cohorts, supporting the similarity between PPCM and DCM. Genetic testing should therefore be considered in women with PPCM.

There is growing evidence that several pathophysiological mechanisms in PPCM converge on a common pathway, which involves inflammation, unbalanced oxidative stress, and the generation of the anti-angiogenic 16 kDa prolactin. The 16 kDa prolactin induces endothelial dysfunction and damage, and subsequently leads to HF. Blocking prolactin with the dopamine D2 receptor agonist bromocriptine has emerged as a potential disease-specific therapy for PPCM.[344,349–352] Additionally, the systemic or local increase in other anti-angiogenic factors, such as the soluble fms-like tyrosine kinase-1 (sFlt-1) receptor, contributes to both local and widespread vascular dysfunction.[353]

7.3. Diagnosis and clinical interventions

Peripartum cardiomyopathy may present as subtle manifestations but most women with PPCM present with acute heart failure (AHF) with severe symptoms (NYHA III/IV). Mild to moderate symptomatic cases of PPCM are often mistaken for physiological changes associated with pregnancy, especially in the post-partum period. Myocarditis is a differential diagnosis and should be excluded by CMR.[354] Women with PPCM and pre-eclampsia had a higher risk of adverse neonatal outcome but also a higher likelihood of left ventricular recovery (LVEF ≥50%).[15] Diagnostic measures in a woman with suspected PPCM should include ECG, NPs, and echocardiography. The management strategy should be discussed within the Pregnancy Heart Team, considering maternal and foetal outcomes (*Figure 7*). Foetal prematurity and low birth weight are common in mothers with PPCM, and children of these mothers have a 3.4 times higher incidence of cardiovascular disease and 5 times higher mortality.[355]

Treatment of AHF caused by PPCM follows the main principles of AHF management during and after pregnancy (*Section 12.6*). Mechanical circulatory support should be considered in women with persistent cardiogenic shock despite medical treatment.[356,357]

Most medications used in the management of HF are foetotoxic and thus contraindicated during pregnancy (i.e. ACE-Is, ARBs, MRAs, and SGLT2 inhibitors).[339] In the post-partum period, full HF treatment

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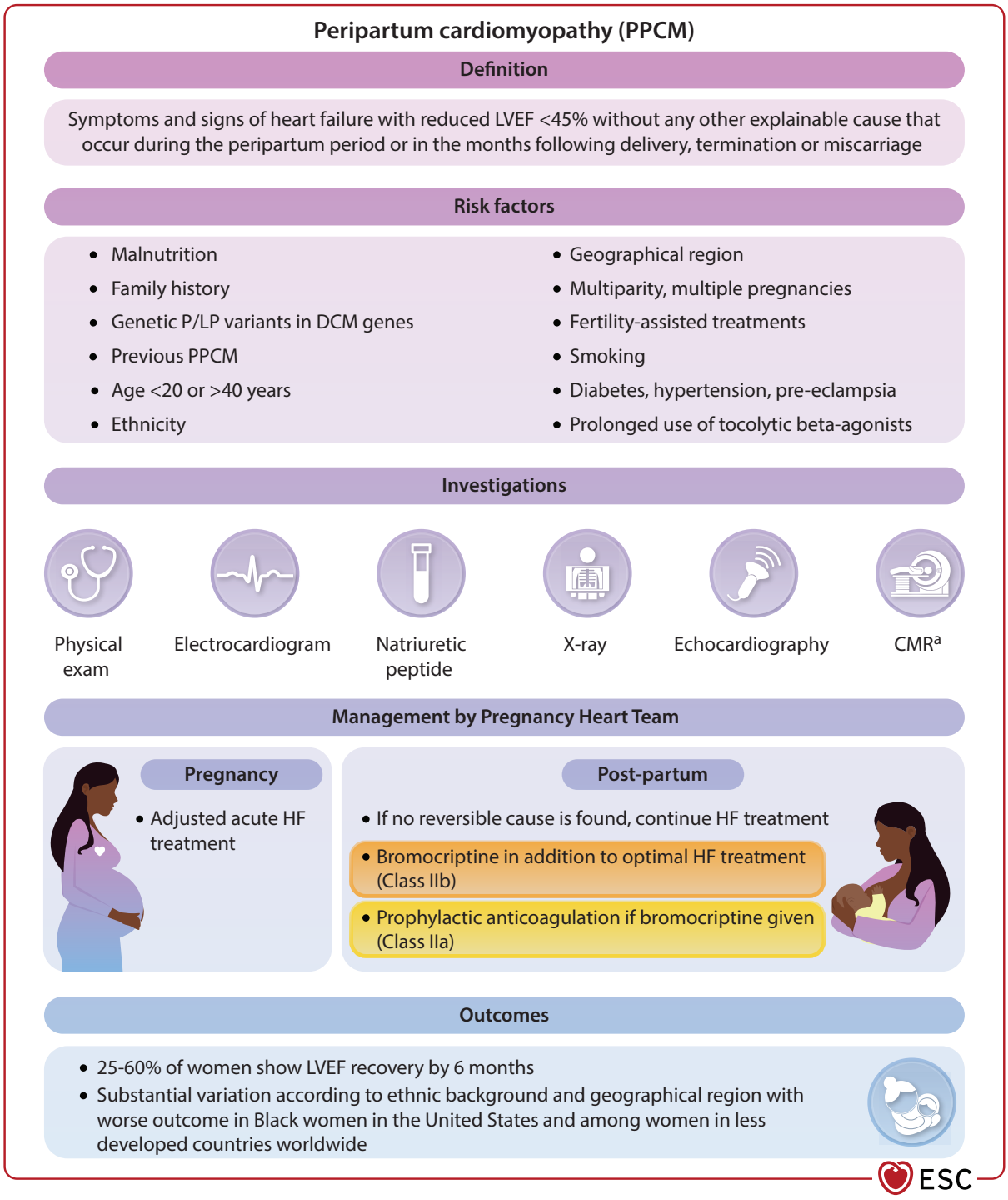
Peripartum cardiomyopathy (PPCM)
Definition
Symptoms and signs of heart failure with reduced LVEF <45% without any other explainable cause that occur during the peripartum period or in the months following delivery, termination or miscarriage
Risk factors
Malnutrition
Family history
Genetic P/LP variants in DCM genes
Previous PPCM
Age <20 or >40 years
Ethnicity
Geographical region
Multiparity, multiple pregnancies
Fertility-assisted treatments
Smoking
Diabetes, hypertension, pre-eclampsia
Prolonged use of tocolytic beta-agonists
Investigations
Physical exam
Electrocardiogram
Natriuretic peptide
X-ray
Echocardiography
CMR[a]
Management by Pregnancy Heart Team
Pregnancy
Adjusted acute HF treatment
Post-partum
If no reversible cause is found, continue HF treatment
Bromocriptine in addition to optimal HF treatment (Class IIb)
Prophylactic anticoagulation if bromocriptine given (Class IIa)
Outcomes
25-60% of women show LVEF recovery by 6 months
Substantial variation according to ethnic background and geographical region with worse outcome in Black women in the United States and among women in less developed countries worldwide
ESC

Figure 7 Risk factors and management of peripartum cardiomyopathy. CMR, cardiac magnetic resonance imaging; HF, heart failure; LVEF, left ventricular ejection fraction; P/LP, pathogenic/likely pathogenic; PPCM, peripartum cardiomyopathy. [a]In specific cases.

can be initiated, except if lactation and breastfeeding are necessary for nutritional reasons, in which case ARBs and SGLT2 inhibitors should be avoided. Spironolactone is considered safe (see *Section 5.2.7* and *Figure 7*).

In addition to HF treatment, the prolactin production suppressing agent bromocriptine may be considered in women with PPCM (*Figure 7*).[358–360] A secondary effect of bromocriptine is stopping lactation, which enables the possibility of full HF treatment of the mother that is not breastfeeding. The downsides of stopping lactation as PPCM treatment include psychological implications for the mother and the source of nutrition for the infant. These considerations indicate that women with moderate and severe HF in PPCM are the preferred candidates for bromocriptine treatment. A recent multi-centre randomized study comparing two different bromocriptine dosages in women

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with severe PPCM (2.5 mg daily for 1 week vs 5 mg daily for 2 weeks followed by 2.5 mg daily for 6 weeks) observed a high LV recovery rate at 6 months. No significant differences were observed between treatment over 1 week and 8 weeks, suggesting that a 1 week addition of bromocriptine to standard heart failure treatment would be beneficial.[358] There are limited data on the use of bromocriptine in pregnant women with PPCM and cardiogenic shock.[339,361] Personalized bromocriptine treatment, with dose adjustments to effectively suppress prolactin, may be a viable therapeutic option in these specific cases. Adding LMWH (in prophylactic doses at a minimum) to bromocriptine should be considered to reduce the thromboembolic risk.[362]

7.4. Outcomes

Risk stratification is crucial to determine the appropriate level of care for women diagnosed with PPCM (*Figure 7*). Key indicators to identify individuals at risk of complications include LVEF <30%, LV end-diastolic diameter >60 mm, biventricular dysfunction, ECG QT interval prolongation, delayed diagnosis, and Middle Eastern or African ethnicity and/or geography.[344] Additional parameters such as age (>40 or <20 years), antepartum diagnosis, haemodynamic parameters at presentation, and cardiac biomarkers can further refine risk stratification.

PPCM may cause ventricular tachyarrhythmias and patients should therefore be monitored.[363,364] As ~50% of women with PPCM recover within 1 year after delivery, a wearable cardioverter defibrillator (WCD) for pregnant women with LVEF <35% at risk of sudden cardiac death may be considered to provide bridging therapy to recovery.[365,366]

Myocardial recovery after PPCM, defined as LVEF >50%, has been shown to occur in 46% (25%–62% according to geographical region) of women at 6 months.[16] Full HF treatment should be given during the first year after complete LV function recovery. Stepwise discontinuation of HF therapy may be considered after 1 year if complete myocardial recovery is achieved, assuming that no genetic predisposition has been identified.[339,367] However, recent data indicated higher risk of LVEF relapse during subsequent pregnancies in PPCM women who had discontinued their HF medication.[368] Left ventricular assist device (LVAD) or heart transplantation have been reported in up to 10% of PPCM cases, with inferior survival rates compared to other age-adjusted heart transplant recipients.[340]

Outcomes after PPCM differ globally.[344] The EURObservational Research Programme's (EORP) PPCM registry reported low mortality rates[369] of 2.4% 1 month after diagnosis.[370] However, the mortality rate at 6 months was 6%, with HF and cardiac arrest as the most frequent causes of death.[16] At 1 year follow-up, death from any cause occurred in 8% of women, with regional variations (Europe 5%, Africa 6%, Asia–Pacific 9%, Middle East 19%; $P < .001$).[17]

Women with a previous PPCM diagnosis face a notably elevated risk of poor outcomes. In the most recent EORP paper following women with subsequent pregnancies after PPCM, risk of maternal mortality was lower than in previous reports, at 2% at 198 days after delivery.[368] More than mild LV dysfunction before a new pregnancy increases the risk of LVEF deterioration, but also women with recovered LV functions remained at risk of relapse.[368] In women planning a new pregnancy after a previous PPCM and with only mild LV dysfunction, stress echo without contraindicated HF medication may be helpful to further stratify risk. Having a good contractile reserve after HF medication has been discontinued may be an encouraging prognostic sign.[371] If planning a new pregnancy after PPCM, discontinuation of beta-blocking therapy may not be advisable, and restarting beta-blocking therapy may be beneficial at a subsequent pregnancy, irrespective of baseline LV systolic function.[368]

Recommendation Table 7 — Recommendations for peripartum cardiomyopathy (see Evidence Table 7)

Recommendations	Class[a]	Level[b]
Counselling for women with PPCM about the risk of recurrence during a subsequent pregnancy and about contraception is recommended in all cases, even after recovery of LV function (LVEF >50%).[355,369]	I	C
Adding at least prophylactic LMWH treatment to bromocriptine treatment in women with PPCM should be considered.[358,362,372,373]	IIa	C
Genetic counselling and testing should be considered in women with PPCM.[60]	IIa	C
When a reversible course of HF is assumed, treatment in accordance with HF guidelines should be considered for at least 12 months after complete LV recovery (normalization of LV volumes and EF).[339,345,368,374]	IIa	C
Bromocriptine treatment may be considered in addition to optimal HF treatment to enhance recovery of LV function in women with PPCM.[358–360,366,375,376]	IIb	B
The use of a WCD may be considered in women with PPCM and LVEF <35%.[377]	IIb	C

EF, ejection fraction; HF, heart failure; LMWH, low-molecular-weight heparin; LV, left ventricle; LVEF, left ventricular ejection fraction; PPCM, peripartum cardiomyopathy; WCD, wearable cardioverter defibrillator.
[a]Class of recommendation.
[b]Level of evidence.

8. Pregnancy in women with aortopathies

Acute arterial dissection during pregnancy occurs in 5.5/100 000 live births, with the aorta being the third most frequent location (19.8%) after coronary artery dissection (38.2%) and vertebral artery dissection (22.9%).[378] A large cohort study reported an aortic dissection rate of 5.5 per million women during pregnancy and post-partum, compared with 1.4 per million during the equivalent period 1 year later.[40] Although rare, acute aortic syndromes carry high foetal and maternal morbidity and mortality risks.[378,379] Arterial dissections occur antenatally in 15%, intrapartum in 23%, and post-partum in 62% of cases.[378] Most pregnancy-related aortic dissections occur in women who are unaware of their underlying aortic disease[380] and events rarely occur in women who have been monitored according to guidelines.[7] The risk of peripartum dissection in more distal aortic segments remains after prior aortic root replacement.[381] The mechanism for dissection during pregnancy is unclear. Given the high post-partum prevalence, haemodynamic changes alone do not fully explain the increased risk and hormonal influences are likely involved.

8.1. Women with heritable thoracic aortic disease

The number of genes associated with heritable thoracic aortic disease (HTAD) is steadily increasing. Although there is clear evidence for an increased risk of aortic dissection in HTAD, recent data from the

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ROPAC III study showed that the aortic dissection incidence rate (3.5%) in pregnant women with HTAD was lower than previously reported.[382] Phenotypes and outcomes between different genes and variants vary, are of clinical importance, and impose differences in management with regard to the extent of imaging, surveillance, and referral for surgery.[62,383] Clinical and genetic entities for which data are available are discussed below and included in *Figure 8*.

8.1.1. Marfan syndrome

Marfan syndrome (MFS) is caused by P/LP variants in *FBN1*. Pregnancy-associated cardiovascular events include aortic and coronary artery dissection as well as rapid aortic growth necessitating surgery.[384] Aortic event rates can reach up to 10%. Although type A dissections mainly occur in undiagnosed women, often with aortic diameters exceeding surgical thresholds,[389] type B dissections remain unpredictable and can occur even after prophylactic root replacement.[390] Women diagnosed earlier in life have a lower risk of dissection.[391] More dissections occur during the post-partum period than during pregnancy or at delivery.[384,392] Event rates and overall maternal mortality are low in women under guidelines-based follow-up.[7,390] Studies have shown stable aortic root diameters during pregnancy in women with diameters between 40 and 45 mm.[384,393] No significant difference in aortic events is noted between ever-pregnant and never-pregnant women,[384,392] but most data come from patients in highly controlled environments.

8.1.2. Loeys–Dietz syndrome

Loeys–Dietz syndrome (LDS) is linked to P/LP variants in six genes: *TGFBR1*, *TGFBR2*, *SMAD2*, *SMAD3*, *TGFB2*, and *TGFB3*. Aortic outcomes in LDS vary by gene and variant,[64] leading to gene-specific recommendations for aortic root surgery thresholds.[62,383,388] Planned pregnancy is a known risk factor, but data on dissection risk during pregnancy, including diameters at dissection, are limited. Cases of type B dissection after aortic root replacement have been reported.[394] A higher incidence of haemorrhagic events, reported in earlier studies,[395] was not corroborated in a recent ROPAC III study.[382] Although pregnancy data for women with LDS are sparse, recent reports suggest favourable maternal and foetal outcomes with appropriate counselling and surveillance.[395] The lack of data about aortic diameters at dissection during pregnancy limits high-level recommendations for prophylactic aortic root surgery thresholds.

8.1.3. Vascular Ehlers–Danlos syndrome

With a reported pregnancy-related mortality rate of 5% and life-threatening vascular events in up to 10% of pregnancies, women with vascular Ehlers–Danlos syndrome undeniably have high-risk pregnancies.[396] Pregnancy-related complications include vascular dissection or rupture, uterine rupture, perineal tears, haemorrhage, and premature birth. However, pregnancy and delivery do not seem to affect overall survival rates in women with vascular Ehlers–Danlos syndrome.[397] The risk may be lower for some women with specific genetic variants, null mutations, and normal vascular imaging.[398,399] Celiprolol is recommended (also in normotensive women), given the very high risk of dissections and the benefit demonstrated in non-pregnant populations.[7] Shared decision-making is crucial for these women. A ROPAC study included four women with vascular Ehlers–Danlos syndrome who experienced pregnancy without adverse maternal events.[7] Based on data from a recent systematic review, caesarean section at 37 weeks should be scheduled to avoid obstetrical complications.[400] Women with vascular Ehlers–Danlos syndrome should be counselled on pregnancy risk and monitored by a Pregnancy Heart Team.

8.1.4. Non-syndromic heritable thoracic aortic diseases

The number of genes linked to non-syndromic heritable thoracic aortic diseases (nsHTAD) is growing, including those rarely associated with extra-aortic features such as *MYLK*, *ACTA2*, *MYH11*, and *PRKG1*.[401,402] For most cases, specific pregnancy management recommendations are limited. Pragmatically, prophylactic surgery in nsHTAD is recommended at a diameter >45 mm. In women with variants in *PRKG1*, certain *ACTA2* variants, or additional risk factors that carry a high dissection risk at small diameters, such as hypertension or family history of dissection at smaller diameters, surgery may be considered at lower diameters (>40 mm).[386]

8.1.5. Aortic disease with no identifiable (likely) pathogenic variant

It is unclear whether young women with known aortic disease in whom genetic screening fails to identify a P/LP variant truly have a lower dissection risk than those with a known variant. The term 'sporadic aneurysm' is discouraged, as an aneurysm may stem from a heritable disorder even without a family history. Recent data from a large cohort of type A aortic dissection patients <30 years of age showed a near dichotomy between HTAD and unknown hypertension as probable dissection causes.[403]

8.2. Turner syndrome

Approximately 50% of women with Turner syndrome (TS) have cardiovascular manifestations, including aortic dilatation, bicuspid aortic valve (BAV), aortic coarctation, elongated aortic arch, and partial abnormal pulmonary venous return.[404,405] All women with TS present a generalized arteriopathy and TS itself is an independent risk factor for thoracic aortic dilatation. Aortic dissection risk (85% type A and 15% type B) increases with increasing diameters and can be reduced by following treatment guidelines.[406–409] Risk factors include hypertension, BAV, and coarctation. In women with TS and an aortic size index (ASI) ≥25 mm/m^2, aortic height index ≥25 mm/m or a z-score >4, the increased dissection risk and the option of surgery before pregnancy should be discussed with the patient and the Pregnancy Heart Team, taking the other risk factors into account.[410–412] Spontaneous pregnancy can occur in women with mosaic TS, but assisted fertility techniques are now more common. Timely cardiovascular evaluation before fertility treatment is very important. Higher rates of adverse events during pregnancy and post-partum have been reported including hypertensive disease, gestational diabetes, haemorrhage, and SGA babies.[413,414] Caesarean section rates of up to 67% have been reported.[414]

8.3. Bicuspid aortic valve disease

Available data for patients with BAV indicate a low risk of aortic events if the aorta is <45 mm. Data on pregnancy in women with diameters of 45–50 mm are limited.[7,415] Recent data on patients with BAV demonstrate a higher risk of dissection in those with a 'root phenotype' compared to those with a primarily ascending aorta involvement.[416,417] When counselling these patients, it is important to note that BAV does not exclude the possibility of nsHTAD.

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8.4. Aortic aneurysms other than root or ascending aorta

Currently there are no recommendations for prophylactic surgery before conceiving except for the aortic root and ascending aorta. Recently published guidelines recommended surgery for the undissected aortic arch, descending and abdominal aorta at 50 mm in patients with Marfan syndrome,[62] but no specific guidelines for management in pregnancy are available.

8.5. Aortic dissection

Data about pregnancy in women with a history of aortic dissection are scarce. ROPAC II and III included 11 and 9 women, respectively, with a previous dissection and reported no maternal mortality. However, two women in ROPAC III had a recurrent aortic event.[7,382] Adverse events are likely low in women with a history of traumatic or iatrogenic dissection and probably also for those with a maximum aortic diameter of <40 mm and documented stable follow-up. Obviously there is a selection bias, as many women with complex aortic disease choose not to have children independent of the risk of progression of aortic disease.

8.6. Management

Women with aortic disease should be managed by a multidisciplinary team with experts from both the Pregnancy Heart Team and the aortic team who are experienced in diagnostic pathways, medical and surgical management of aortopathies in the ante-, peri-, and post-partum periods. Institutional protocols for the management of pregnancy-related aortic events should be available and a shared-decision model needs to be applied. Imaging of the entire aorta (CT or CMR) in women with known or suspected aortic disease is recommended and can reasonably be performed for most clinical scenarios within the 6 months prior to pregnancy. CMR without gadolinium is recommended in pregnant women with known aortopathy, without pre-pregnancy imaging. In all women with aortic disease, strict blood pressure control is recommended. Specific target values for this specific situation have not been studied—in the general population it is recommended that treated systolic blood pressure values be targeted to 120–129 mmHg to reduce CVD, provided the treatment is well tolerated.[388,418] In women with genetic aortopathies, treatment with beta-blockers throughout pregnancy should be considered with foetal growth monitoring. Women who used beta-blockers

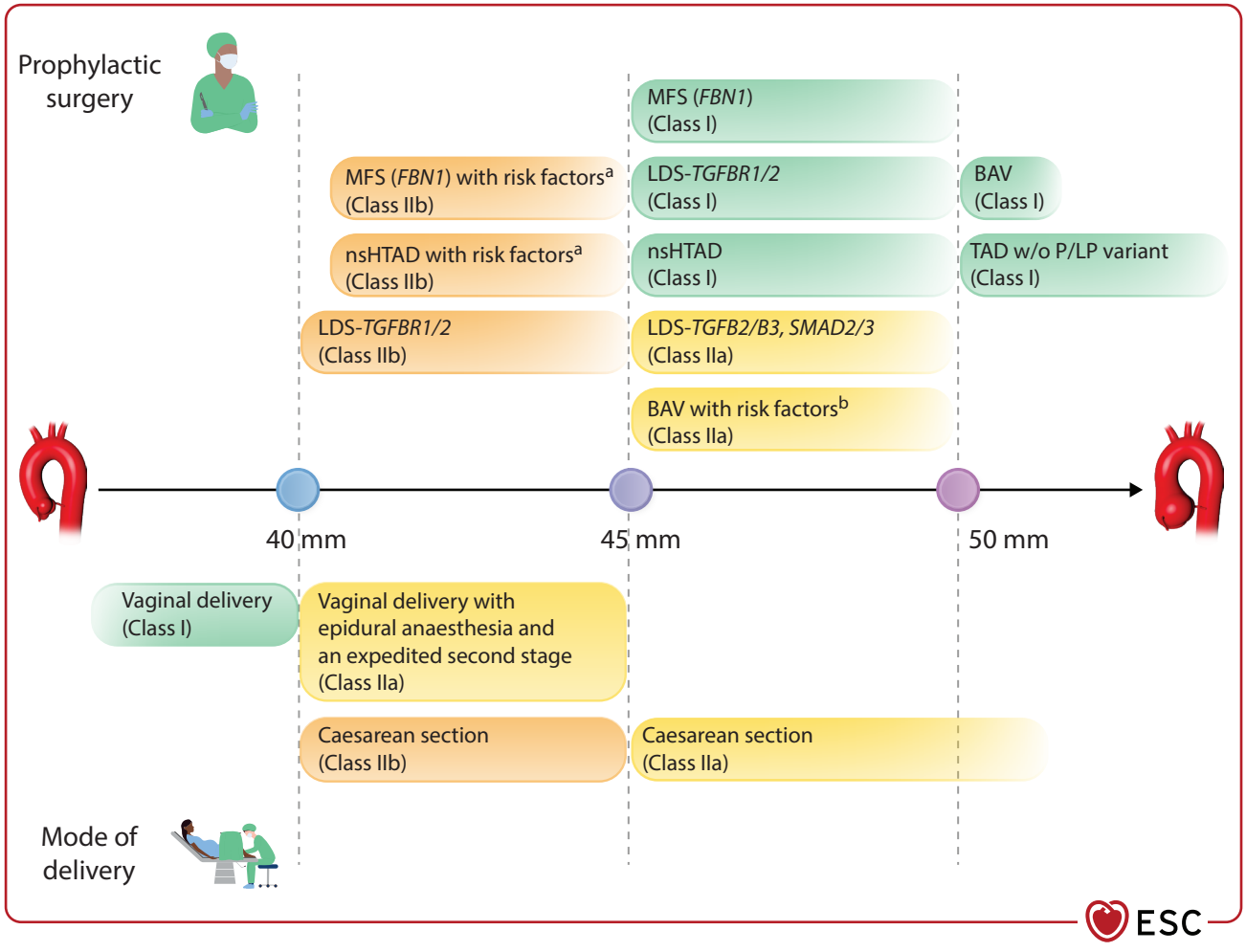
Prophylactic surgery
MFS (FBN1) (Class I)
MFS (FBN1) with risk factors[a] (Class IIb)
LDS-TGFBR1/2 (Class I)
BAV (Class I)
nsHTAD with risk factors[a] (Class IIb)
nsHTAD (Class I)
TAD w/o P/LP variant (Class I)
LDS-TGFBR1/2 (Class IIb)
LDS-TGFB2/B3, SMAD2/3 (Class IIa)
BAV with risk factors[b] (Class IIa)
40 mm
45 mm
50 mm
Vaginal delivery (Class I)
Vaginal delivery with epidural anaesthesia and an expedited second stage (Class IIa)
Caesarean section (Class IIb)
Caesarean section (Class IIa)
Mode of delivery
ESC

Figure 8 Thresholds for prophylactic surgical treatment prior to pregnancy of aortic root/ascending aneurysm (above the line) and recommended mode of delivery according to aortic diameter (below the line). BAV, bicuspid aortic valve; LDS, Loeys–Dietz syndrome; MFS, Marfan syndrome, nsHTAD, non-syndromic heritable thoracic aortic disease; P/LP, pathogenic/likely pathogenic; TAD, thoracic aortic disease; w/o, without. [a]Risk factors: family history of dissection, rapid aortic growth (≥3 mm/year), uncontrolled hypertension. [b]Root phenotype to be added to the other risk factors. Based on Narula *et al.*,[384] Wallace *et al.*,[385] Regalado *et al.*,[386] Jondeau *et al.*,[387] Mazzolai *et al.*,[388] and Isselbacher *et al.*[62].

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throughout pregnancy in the ROPAC III study gave birth to infants with a significantly lower birth weight than women who did not.[382] Specific thresholds for pre-conception aortic root surgery are illustrated in *Figure 8*.

The mode of delivery needs to be informed by the patient's history, presence and type of gene/variant, and aortic diameter. The primary aim of intrapartum management in women with aortic disease is to reduce the cardiovascular stress of labour and delivery. Caesarean section may be considered in women with a maximum diameter of >45 mm and those with a history of dissection. In women with an aortic diameter <40 mm, vaginal delivery is recommended. In women with MFS, LDS, and vascular Ehlers–Danlos syndrome, pre-delivery anaesthesiology consultation is recommended to consider precautions for dural ectasia and scoliosis (including possible previous surgery).[419] In general, an individualized approach is favoured.[383] Given the peak risk of a dissection at day 6 post-partum, it may be appropriate for women to stay in hospital for 1 week post-partum.[420]

8.7. Cardiac surgery during pregnancy

Prophylactic cardiac surgery in women who are planning a pregnancy is far more common than urgent or emergency surgery during pregnancy. Aortic surgery is the most frequently performed procedure during pregnancy, followed by surgery for aortic and mitral valve disease. Key pregnancy-related pathologies include prosthetic valve thrombosis and endocarditis. The decision to perform surgery with extracorporeal circulation and cardioplegic arrest during pregnancy is highly individualized with no specific guidelines available. A recent meta-analysis[421] reported a maternal mortality rate of 7.3% consistent across trimesters, and no difference in maternal mortality if caesarean section was performed. Overall foetal mortality was 26.5%, lowest during third trimester surgeries (10.3%). Caesarean section before surgery significantly reduces foetal mortality. The decision to perform a caesarean section prior to surgery should be based on foetal viability and the level of medical care available rather than on a fixed gestational age. This is particularly crucial in acute type A aortic dissection. Hypothermic distal circulatory arrest increases foetal mortality.[422]

Recommendation Table 8 — Recommendations for aortopathies, cardiac surgery, and pregnancy

Recommendations	Class[a]	Level[b]
Counselling		
It is recommended that women with aortic disease have counselling about the risk of aortic dissection in pregnancy and the post-partum period.[404,423]	I	C
It is recommended that women with a history of aortic dissection or -surgery have pre-pregnancy counselling about the high risk by an extended Pregnancy Heart Team[c] considering the presence and type of genetic variant, aortic morphology, growth rate, and aetiology of aortic dissection.[7,381,424]	I	C
It is recommended that women with vascular Ehlers–Danlos syndrome wishing to become pregnant are counselled regarding the very high risk of pregnancy-related adverse events by a multidisciplinary team, considering family history, genetic variant, and previous vascular events.[7,396,397,425]	I	C
Imaging		
Imaging of the entire aorta[d] (CT or CMR) is recommended before pregnancy in women with known or suspected aortic disease.[390,424,426]	I	C
In women with aortic dilatation related to BAV, imaging (with TTE, and CMR/CT if needed) of the aortic root, ascending aorta, and descending aorta (to rule out coarctation) is recommended before pregnancy.[427,428]	I	C
In women with low-risk aortic disease (mWHO 2.0 classes II and II–III), one-time echocardiographic imaging between 20 and 30 weeks of gestation and imaging at 6 months post-partum is recommended.[393,429]	I	C
In women with moderate to high-risk aortic disease (mWHO 2.0 classes III and IV), repeated echocardiographic imaging every 4–12 weeks (depending on diagnosis and severity of dilatation) is recommended during pregnancy and until 6 months post-partum.[393,429,430]	I	C
CMR (without gadolinium) imaging of the entire aorta is recommended in pregnant women at risk of or with known aortic dilatation who have not had recent pre-pregnancy cross sectional imaging.[135]	I	C
Treatment—medical		
When a woman with known aortic dilatation, history of dissection, or P/LP variant associated with aortic disease becomes pregnant, strict and individualized BP control is recommended.[431,432]	I	C
Beta-blocker therapy[e] throughout pregnancy and in the post-partum period is recommended in women with MFS and other HTADs.[433]	I	C
Celiprolol is recommended in women with vascular Ehlers–Danlos syndrome during pregnancy and lactation.[434]	I	C
Treatment—intervention/surgical		
It is recommended that indications for pre-pregnancy aortic root and/or ascending aortic surgery are guided by aortic morphology, underlying pathology, family history, genetic variant, previous vascular events, and patient's preference.[62,383]	I	C
It is recommended that centres managing pregnancies in women with moderate to high-risk aortic disease (mWHO 2.0 class III/IV) can provide cardiovascular surgery in case of peripartum adverse events.[435,436]	I	C
Specific conditions		
In women with MFS and aortic root diameters >45 mm, surgery before pregnancy is recommended.[380,424]	I	C
In women with LDS with P/LP variants in *TGFBR1*, *TGFBR2*, and aortic root diameters ≥45 mm, surgery before pregnancy is recommended.[437–439]	I	C

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In women with nsHTAD with P/LP variants in *MYH11*, *ACTA2*, *PRKG1*, or *MYLK*, and aortic root diameters ≥45 mm, surgery before pregnancy is recommended.[385,386,440]	I	C
In women with BAV and aortic root or ascending aortic diameter ≥50 mm, surgery before pregnancy is recommended.[441,442]	I	C
In women without an identifiable P/LP variant with aortic root or ascending aortic diameters ≥50 mm, surgery before pregnancy is recommended.	I	C
In women with HTAD and aortic arch, descending aortic, or abdominal aortic diameters ≥50 mm, surgery before pregnancy should be considered.[392,443]	IIa	C
In women with LDS with P/LP variants in *TGFB2*, *TGFB3*, *SMAD2*, and *SMAD3*, and aortic root diameters ≥45 mm, surgery before pregnancy should be considered.[437–439]	IIa	C
In women with BAV and root phenotype or family history of aortic aneurysm or dissection, surgery before pregnancy should be considered if the aorta is ≥45 mm.	IIa	C
In women without an identifiable P/LP variant with aortic root or ascending aortic aneurysm ≥45 mm, surgery before pregnancy should be considered in the presence of a family history of aortic aneurysm, aortic dissection, uncontrolled arterial hypertension, or on patient's preference.	IIa	C
In women with MFS and aortic root diameters between 40 and 45 mm, surgery before pregnancy may be considered if risk factors (growth >3 mm/year, family history of aortic dissection) are present.[444]	IIb	C
In women with LDS with P/LP variants in *TGFBR1* or *TGFBR2* and aortic root diameters ≥40 mm, surgery before pregnancy may be considered.[387,437,445,446]	IIb	C
In women with nsHTAD and aortic root diameters ≥40–44 mm, surgery before pregnancy may be considered depending on the genetic variant, family history, and aortic growth rate.[385,386,440]	IIb	C
Delivery		
In women with an aorta <40 mm, vaginal delivery is recommended.[163]	I	C
In women with vascular Ehlers–Danlos syndrome, caesarean section at 37 weeks is recommended for obstetrical reasons.[400]	I	C
In women with an aorta 40–45 mm, vaginal delivery with epidural anaesthesia and an expedited second stage should be considered.	IIa	C
In women with an aorta ≥45 mm, caesarean section should be considered.	IIa	C
In women with acute, subacute, or chronic aortic dissection, caesarean section should be considered.	IIa	C
In women with an aorta 40–45 mm, caesarean section may be considered.	IIb	C
The use of ergometrine post-delivery is not recommended in women with aortopathy.	III	C
Cardiac surgery during pregnancy		
Delivery before cardiac surgery should be considered as soon as the foetus is viable, taking gestational age, comorbidities, and the available level of neonatal care into account.[163,421,435,447]	IIa	C
Cardiac surgery may be considered during pregnancy when conservative and medical therapy has failed, and in situations that threaten the mother's life or that are not amenable to percutaneous treatment.	IIb	C

BAV, bicuspid aortic valve; BP, blood pressure; CMR, cardiovascular magnetic resonance imaging; CT, computed tomography; HTAD, heritable thoracic aortic disease; LDS, Loeys–Dietz syndrome; MFS, Marfan syndrome; mWHO, modified World Health Organization; nsHTAD, non-syndromic heritable thoracic aortic disease; P/LP, pathogenic/likely pathogenic; TTE, transthoracic echocardiogram.
[a]Class of recommendation.
[b]Level of evidence.
[c]Extended Pregnancy Heart Team: regular team + multidisciplinary aortic team—see also *Section 4.1*.
[d]In women with vascular Ehlers–Danlos syndrome and LDS, imaging should encompass the entire aorta, including supra-aortic vessels as well as iliac and femoral arteries.
[e]See *Section 5.2.9* for beta-blocker choice.

9. Pregnancy in women with known congenital heart disease

Congenital heart disease is present in 0.8%–0.9% of live births, with significant geographical variation.[448,449] Nowadays, most children born with congenital heart disease reach fertile age, even those with complex lesions, making adult congenital heart disease (ACHD) one of the most frequent CVDs during pregnancy.[450] Discussions about family planning, contraception, pregnancy risk (mWHO 2.0—see *Table 6*), and life expectancy are essential and should start early, preferably during the transition to adult life.[2]

In the prospective ROPAC registry, most women with ACHD (n = 3295) had a relatively favourable pregnancy outcome, with an overall mortality rate of 0.2% and trends improving from 2007 to 2017.[2] Heart failure rates were low in ROPAC, with differences according to disease complexity (13% in severe, 5%–6% in less-complex lesions). Arrhythmia rates were low overall (2%). Women with uncorrected ACHD more often have maternal and foetal complications.[451] Women with ACHD experiencing complications during pregnancy and post-partum may also be at higher risk of late cardiac events.[451]

Pre-pregnancy evaluation should at least include routine blood tests, ECG, TTE, and cardiopulmonary exercise testing. As mentioned in *Section 4*, the increased transmission risk should be discussed, and genetic counselling should be offered.[452] The level and timing of the pre-pregnancy cardiovascular evaluation and follow-up during pregnancy depend on the mWHO 2.0 class (see *Table 6*), taking the anatomical and functional status into account.

Optimization of cardiac status and any comorbidities should take place prior to pregnancy. This includes guideline-directed elective surgery or/and

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intervention of significant haemodynamic lesions (native or residual),[21] as well as optimizing medical therapy and healthy lifestyle choices.

The timing and mode of delivery should be decided by the Pregnancy Heart Team, for all women with a condition of mWHO 2.0 class II–III or above. In general, vaginal delivery is the preferred delivery mode in women with ACHD.[163] Post-partum monitoring should be individualized based on the woman's underlying ACHD, risk or presence of arrhythmias and/or HF, and the course during pregnancy and delivery. High-risk women or those with HF symptoms during pregnancy or delivery should be considered for intensive (cardiac) care admission for haemodynamic monitoring.[183] For more details about managing the various delivery stages, including post-partum, see *Sections 4.5* and *4.6.1*.

A summary of relevant disease-specific considerations, maternal and foetal complications, monitoring, and management during pregnancy are listed in *Table 12*.

Table 12 Risks, monitoring, and management during pregnancy and delivery in women with congenital heart disease

ACHD	Maternal Risk	Obstetric and foetal risk	Monitoring	Pregnancy management and delivery
Left ventricular outflow tract obstruction (LVOTO)				
Coarctation of the aorta	• ↑ Complication risk if residual obstruction (gradient >20 mmHg, aortic lumen <12 mm), clinical signs of HF, LVEF <40%, NYHA class>1[9] • ↑ Risk of aortic dissection (if aneurysm present) • Uncontrolled hypertension[9]	• ↑ Miscarriage rate[453] • Pre-term birth and low birth weight in 9%[9]	• Close BP monitoring —also early post-partum • Pre-pregnancy CMR and treatment of residual lesions[454]	• Treat hypertension[a] • Consider bed rest, hospital admission and stenting in case of severe symptomatic (re)coarctation (including refractory hypertension or maternal/foetal compromise)[455] • Vaginal delivery preferred unless aneurysm, HF, severe hypertension
Subvalvular, valvular and supravalvular aortic stenosis: see *Section 12.5.1*[b] and *Section 8.3*[c] for BAV related aortic disease. Women with serial left heart obstructive lesions have higher maternal cardiovascular event rates.[456]				
Shunt lesions				
ASD	• Low risk in (un)repaired ASD (if no PAH)[457] *Unrepaired ASD:* • ↑ Risk of arrhythmia (4%)[457] • Paradoxical embolism (2%–5%)[457,458]	• Rare[457] • Unrepaired ASD [457,458]: • SGA (21%) • Foetal/perinatal mortality (2%–3%) • Pre-eclampsia (7%)	*Unrepaired and uncomplicated ASD:* consider TTE at 28–32 weeks[457]	• Large and/or haemodynamically significant ASD: closure pre-pregnancy[21] • Unrepaired ASD: • Consider ASA or prophylactic LMWH[d] for paradoxical embolism prevention • Consider device closure in pregnancy only for recurrent stroke on medical therapy
VSD and patent ductus arteriosus	Low risk in small or repaired lesions with normal LV and no PAH[e]	No evidence for ↑ risk	*Unrepaired and uncomplicated VSD:* consider TTE at 28–32 weeks	Vaginal delivery is preferred
AVSD	• Low risk in repaired AVSD without significant residual lesions • Arrhythmia and ↑ AV valve regurgitation and HF if residual left AV valve regurgitation[459,460] • ↑ Paradoxical emboli risk in unoperated (partial) AVSD	• Offspring mortality in 6% primarily due to recurrence of the congenital heart disease[459]	*Unrepaired and uncomplicated AVSD:* consider TTE at 28–32 weeks ↑ FU frequency in significant valve regurgitation, PAH, ↓ ventricular function, or ↑ NYHA class[461]	• Residual shunt: see ASD and/or VSD • ↑ AV valve regurgitation and/or HF[f] • PAH[e] • Delivery: see ASD and VSD
Pulmonary valve and RVOT disease				
RVOTO/PV stenosis	• Mild to moderate: low risk • *Severe*: RV failure and arrhythmia[21]	• Very low complication risk[462,463]	• Mild to moderate: • TTE at 28–32 weeks • *Severe stenosis:*	*Pre-pregnancy severe RVOTO (Doppler peak gradient>64 mmHg) or/and any signs of right HF:*

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			• (Bi)monthly TTE (focused on RV function)	• Intervention (at any level of the RVOT)[21] *Severe symptomatic PV stenosis (not responding to bed rest and conservative management):* • Consider transcatheter balloon valvotomy[21] Consider caesarean section in severe RVOTO/PV stenosis[463]
PV regurgitation	• ↑ Risk when impaired RV function[462]	• Premature birth[463]	Bimonthly TTE if severe PV regurgitation and ↓ RV function	
Post pulmonary valve replacement (surgical or transcatheter without severe stenosis/regurgitation)	• Low risk[463–466]	• Very low complication risk[462,463]	TTE at 28–32 weeks	Vaginal delivery is preferred
Repaired TOF				
	• Low risk if no residual lesions[467] • ↑ Risk of arrhythmia and HF (7%–10%) if pulmonary regurgitation, ↓ RV function, severe RVOTO[467,468]	• 15% risk of foetal and obstetric complications, mainly pre-term delivery and low birth weight[467] • Low foetal mortality (0.7%)[467] • Recurrence risk in the offspring in *22q11* deletion syndrome: 50%	• First trimester and at 28–32 weeks FU with TTE (increase FU depending on functional status)	• RV failure management: Bed rest and diuretics • Arrhythmia management[g] • Severe PV stenosis/regurgitation: see above • Vaginal delivery is preferred Consider caesarean section in severe RVOTO/PV stenosis
Ebstein anomaly				
	• Overall MACE rate in ROPAC 9.9% • Low risk in mild/ moderate Ebstein • (Very) high risk when pre-pregnancy HF, cyanosis due to atrial shunt • ↑ Arrhythmia risk due to accessory pathways[469]	• Foetal risk is related to ↓ maternal CO and cyanosis[3,469]: • Miscarriage • Pre-term birth (20%–24%) • Neonatal death (3%) • PPH • Recurrence risk (5%)	• *Mild to moderate*: baseline and 28–32 weeks assessment with TTE and ECG • *Severe*: monthly/ bimonthly TTE & ECG • Monitor for arrhythmias if palpitations	• Severe tricuspid regurgitation with HF can usually be managed medically • Treat arrhythmias promptly[g] • Appropriate pre-pregnancy counselling about very high risk of MACE when HF and/or cyanosis
Transposition of the great arteries				
TGA after atrial switch (Mustard or Senning) and CCTGA	• High pregnancy risk—MACE rate up to 28% in retrospective series[470]—lower risk in ROPAC[6]: Atrial and ventricular arrhythmias in 6.7%, HF in 10% • Atrial arrhythmias often poorly tolerated • Baffle leaks may lead to desaturation and paradoxical embolism[471] • Predictors of MACE: symptoms of HF before pregnancy and systemic RV EF <40%[6]	• Foetal risks associated with maternal CO and saturation • Pre-term birth (21%) • Low birth weight (18%–21%)[6,472,473] • Rare foetal and neonatal death (1%)[474] • PPH (7%)[6]	• According to anatomical and functional status: TTE every 1–3 months and serial NP • Holter monitoring if palpitations	• Pre-pregnancy counselling about very high risk if NYHA class III/IV, systemic RV EF <40%, more than moderate tricuspid regurgitation, or treated HF[472,473,475] • Treat HF primarily with medical therapy[h] • Promptly treat arrhythmia[g] • Consider prolonged post-partum monitoring (48–72 h) and early post-partum FU given the ↑ risk of post-partum HF No clear evidence for long-term deterioration or ↑ cardiovascular events associated with pregnancy[6,476]
TGA with arterial switch	• Low risk • Ventricular arrhythmias (2.5%–7%), HF (2%–4%)[8]	• Low rate of prematurity or foetal loss[474,477]	• Consider TTE at 20 weeks • Intensify if ↓ ventricular function, ↑ aortic	• Vaginal delivery is preferred • Surgery before pregnancy when the neo-aortic root is >55 mm[21,478] or if severe AR[f]

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			regurgitation and ↑ aortic dilatation	
Single ventricle physiology palliated with Fontan circulation				
	High pregnancy risk *CV events*[479]: • Supraventricular arrhythmias (8%–11%) • HF (4%–14%) *Risk factors:*[479] • Oxygen saturations <85% • ↓ Ventricular function • Arrhythmias • Significant valvular disease • NYHA class III/IV • FALD	↑ *Very high foetal complication risk:*[479–483] • Low live birth rate (56%) • Miscarriages (45%–54%) • SGA (20%–55%) • Premature birth (59%–72%) • Neonatal death (5%–18%) *Obstetric risk:* • Hypertension (14%) • PPH (13%)	• According to anatomical and functional status: TTE every 1–3 months and serial NP[484] • FU in specialized ACHD centre	• Pre-pregnancy counselling about very high risk especially if any risk factor (see maternal risk) • Pregnancy may be well tolerated and successful in a subset of women with single ventricle and Fontan circulation without complications[479,480,485–487] • ASA and/or LMWH (depending on the presence of complications) should be considered in shared decision[484] • Atrial tachyarrhythmias should be promptly treated with cardioversion[i] *Labour/delivery with preload dependent circulation:*[461] • Epidural with slow titration • Labour in left lateral decubitus position • Low thresholds for assisted second stage (↓ Valsalva duration) • i.v. air filter (if fenestration or significant venovenous collaterals)
Unrepaired cyanotic ACHD (without pulmonary hypertension)				
	HF, thrombosis, arrhythmia and endocarditis in ≥15%[488]	Degree of maternal hypoxaemia is the most important predictor of foetal outcome: 10% foetal loss if resting maternal blood saturation >90%, chance of a live birth 12% if maternal oxygen saturation<85%[489]	FU in expert centre	• Pre-pregnancy counselling about very high risk especially if maternal resting saturation <85%[489] • i.v. air filter

ACHD, adult congenital heart disease; ASA, acetylsalicylic acid; ASD, atrial septal defect; AV, atrioventricular; AVSD, atrioventricular septal defect; BAV, bicuspid aortic valve; BP, blood pressure; CCTGA, congenitally corrected transposition of the great arteries; CMR, cardiovascular magnetic resonance; CO, cardiac output; CV, cardiovascular; ECG, electrocardiogram; EF, ejection fraction; FALD, Fontan-associated liver disease; FU, follow-up; HF, heart failure; i.v., intravenous; LMWH, low-molecular-weight heparin; LV, left ventricle; LVEF, left ventricular ejection fraction; LVOTO, left ventricular outflow tract obstruction; MACE: major adverse cardiovascular events; NP, natriuretic peptide; NYHA, New York Heart Association; PAH, pulmonary arterial hypertension; PPH, post-partum haemorrhage; PV, pulmonary valve; ROPAC, Registry of Pregnancy and Cardiac Disease; RV, right ventricle, RVOTO, right ventricle outflow tract obstruction; SGA, small for gestational age; TGA, transposition of the great arteries; TOF, tetralogy of Fallot; TTE, transthoracic echocardiogram; VSD, ventricle septal defect.

↑ increase ↓ decrease.

[a]Refer to *Section 12.3* Hypertensive disorders.

[b]Refer to *Section 12.5.1* Stenotic native valve lesions.

[c]Refer to *Section 8.3* Bicuspid aortic valve disease.

[d]Refer to *Section 5* Drugs during pregnancy and lactation.

[e]Refer to *Section 10* Pregnancy in women with pulmonary arterial hypertension.

[f]Refer to *Section 12.5.2* Regurgitant native valve lesions.

[g]Refer to *Section 12.4* Arrhythmias.

[h]Refer to *Section 12.6* Heart failure.

[i]Refer to *Section 12.4.3* Cardioversion, ablation, and device implantation and implantable cardioverter defibrillator management.

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Recommendation Table 9 — Recommendations for congenital heart disease and pregnancy (see Evidence Table 8)

Recommendations	Class[a]	Level[b]
Vaginal delivery is recommended in most women with ACHD.[161,163]	I	B
It is recommended that all women with Fontan circulation who wish to become pregnant receive counselling from the Pregnancy Heart Team regarding the high risk of pregnancy-related adverse events.[479,480,484,486,487]	I	C
It is recommended that women with a systemic RV (Mustard/Senning or congenitally corrected TGA), in NYHA class III/IV, systemic ventricular dysfunction (EF <40%), or severe TR wishing to become pregnant are counselled by the Pregnancy Heart Team regarding the high risk of pregnancy-related adverse events.[473–475]	I	C
In women with significant haemodynamic lesions, discussion about guideline-directed interventions is recommended prior to pregnancy.	I	C

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ACHD, adult congenital heart disease; EF, ejection fraction; NYHA, New York Heart Association; RV, right ventricle; TGA, transposition of the great arteries; TR, tricuspid regurgitation.
[a]Class of recommendation.
[b]Level of evidence.

10. Pregnancy in women with pulmonary arterial hypertension

Pulmonary hypertension (PH) is defined as a mean pulmonary arterial pressure >20 mmHg, derived from invasive right-heart catheterization, and is classified by aetiology and pathophysiology.[490] Pulmonary arterial hypertension (PAH) is pre-capillary PH characterized by a pulmonary vascular resistance >2 Wood units and pulmonary arterial wedge pressure ≤15 mmHg. Untreated, idiopathic PAH results in death within a median of 2.8 years, but with PAH therapies, median survival extends to over 7 years.[491,492] There is a preponderance of females in the incidence of PAH, and this includes women of childbearing age. The first clinical manifestations may be seen in pregnancy.[493]

Women with PAH should be managed by a Pregnancy Heart Team, and a PH expert experienced in diagnostic pathways, medical treatment, and anticoagulation management from the ante- to the peri- and post-partum period.

10.1. Pre-existing pulmonary arterial hypertension

In women with PAH, maternal and foetal outcomes vary according to the PAH subset. With improved treatment of PAH and a multidisciplinary approach during pregnancy and the peri-partum period, maternal mortality has declined but remains high, ranging from 11% to 25%.[2,490,494,495]

10.1.1. Maternal and foetal risk

Although there is no safe cut-off for elevated pulmonary artery pressure and risk, pregnancies with mild PAH and vasoreactive PAH seem to have better maternal and foetal outcomes than those with moderate to severe PAH.[141,496–498] Women with severe PAH, non-vasoreactive idiopathic PAH, and Eisenmenger syndrome have the highest risk of maternal and foetal mortality.[496,497,499–502] As the clinical course of PAH during pregnancy remains associated with unforeseeable risks and pregnancy may accelerate PAH progression, all women with PAH[496] wishing to become pregnant should be counselled by a multidisciplinary team regarding the very high risk of pregnancy-related adverse events.

Women with Eisenmenger syndrome are unlikely to tolerate pregnancy due to additional risks of RV failure and paradoxical emboli. Chronic cyanosis may worsen due to systemic vasodilatation during pregnancy, an increased right-to-left shunt, and decreased pulmonary blood flow.

Foetal and neonatal mortality risk is high in women with PAH, mainly related to pre-term delivery, reduced maternal CO, and/or hypoxaemia.[503] Miscarriage is common. If oxygen saturation is >90%, there is usually a better foetal outcome (10% foetal loss). If oxygen saturation is <85%, miscarriage, IUGR, prematurity, and foetal death are common (live birth rate of only 12%).[504,505]

10.1.2. Counselling and contraception

Women of childbearing potential with PAH should be counselled at the time of diagnosis about the very high risk and uncertainties associated with becoming pregnant. Clear advice against becoming pregnant, including referral for psychological support if needed, and clear contraceptive advice are required, taking into account the woman's individual needs. Reduced efficacy of hormonal contraceptives should be carefully considered and discussed with women treated with ERAs, as well as the addition of barrier methods for contraception.[490,493]

10.1.3. Management during pregnancy

When pregnancy occurs, termination should be discussed. Rigorous planning with the optimization of targeted PAH therapies and close monitoring are key in managing women with PAH who wish to continue with the pregnancy after appropriate counselling on the high maternal and foetal risks.

Bed rest may be required in symptomatic women, and it may be appropriate to avoid additional risk factors (such as air travel). Right-heart catheterization can be performed to assist management in women showing deterioration.

Diuretics may be needed in women with HF, and iron deficiency should be treated.[43] It is recommended to stop ERAs, riociguat, and selexipag because of potential or unknown teratogenicity.[43,490] PAH therapies that can be used during pregnancy include phosphodiesterase-5 inhibitors and prostacyclin analogues. Sildenafil is used in the vast majority of women and is combined with a prostacyclin analogue depending on the disease severity. The subset of women with true vasodilator responsiveness who are well controlled on CCB therapy should continue taking this during pregnancy.[43,490,506]

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In women with Eisenmenger syndrome, caution is warranted when administering drugs that may lead to sudden systemic vasodilation or a risk of paradoxical air embolism (i.v. line filters should be used for i.v. therapies). Thromboembolism is a major risk, and anticoagulation regimens (type and dosage) should be considered individually, balancing the risk of bleeding vs VTE at each stage of pregnancy. LMWH is most commonly used. Regular follow-up is advisable, initially every 2–4 weeks and then weekly in the third trimester. Women need to be monitored for increasing hypoxaemia and symptoms of HF, including breathlessness, syncope, and congestion. Regular echocardiography and blood testing, including NP levels, can provide evidence of HF where appropriate.

10.2. New diagnosis in pregnancy

The usual diagnostic algorithm as per the 2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension should be followed in a pregnant woman with suspected PAH. Right-heart catheterization should be considered if there is diagnostic uncertainty in identifying *de novo* PAH in pregnancy and to assist with important therapeutic decisions.[490] If this is required, it should be performed in a specialized centre. An individualized approach is required for starting PAH therapies. Many centres start therapy with oral sildenafil. Close follow-up by an experienced Pregnancy Heart Team with a PH expert is needed and prompt escalation of PAH therapy, usually with i.v. epoprostenol, is indicated depending on the disease severity.

10.3. Delivery in women with pulmonary arterial hypertension

A detailed delivery plan, including the optimal mode and timing of delivery, should be provided on a timely basis by the Pregnancy Heart Team with a PH expert. Early delivery, with careful tracking of foetal growth, is often required in women with progressive, decompensated HF not responding to PAH therapies and to reduce the risk of an unplanned birth outside an expert centre.[245] Therapeutic LMWH should be stopped 24 h prior to any mode of delivery to reduce the risk of maternal haemorrhage. Caesarean section providing for a more controlled delivery may be preferred over vaginal delivery.[494,507] Regional anaesthesia is usually favoured over general anaesthesia.

10.3.1. Peri- and post-partum monitoring

Due to the rapid changes in haemodynamics, the post-partum period is particularly high risk, with the majority of maternal mortality occurring after delivery.[245] During delivery and post-delivery, women should be monitored in the intensive care setting with ECG, pulse oximetry, CO monitoring, meticulous fluid balance with central venous pressure monitoring, and optimization of RV function all important determinants of a good outcome. Women remain at high risk for many months after delivery, and individualized counselling is required to discuss the need for ongoing therapies and the avoidance of future pregnancies. In women with severe PAH and those who had complications during pregnancy and/or delivery, optimization of PAH therapies should be prioritized over lactation.

Recommendation Table 10 — Recommendations for pulmonary arterial hypertension and pregnancy

Recommendations	Class[a]	Level[b]
It is recommended that women of childbearing potential with PAH wishing to become pregnant are counselled by a multidisciplinary team regarding the very high risk of pregnancy-related adverse events, encouraging a shared decision-making process about whether to become pregnant.[490]	I	C
It is recommended to provide clear contraceptive advice to women of childbearing potential with PAH.[490]	I	C
For women with PAH requiring pregnancy termination, it is recommended to perform this in PH centres.[490]	I	C
Right-heart catheterization should be considered during pregnancy if there is diagnostic uncertainty or to assist with important therapeutic decisions.[490]	IIa	C
Endothelin receptor antagonists, riociguat, and selexipag are not recommended during pregnancy.[490,508,509]	III	C

PAH, pulmonary arterial hypertension; PH, pulmonary hypertension.
[a]Class of recommendation.
[b]Level of evidence.

11. Venous thromboembolism in pregnancy and post-partum

11.1. Epidemiology and maternal risk

Venous thromboembolism includes PE and DVT. The pooled incidence of pregnancy-related VTE (including post-partum) is 1.2 per 1000 deliveries[510], and it is a major cause of pregnancy-related morbidity and mortality.[510] Pregnancy-related VTE has a fatality rate of 0.68% and a recurrence rate (during pregnancy and post-partum) of 4.27%, which is higher post-partum.[510] A documented assessment of VTE risk factors is recommended before or in early pregnancy. The VTE risk is highest in the third trimester and in the first 6 weeks post-partum.[510] Mortality in pregnancy-related VTE is associated with CVD, hypertension, twin gestation, pre-term birth, caesarean section, transfusion, and black ethnicity.[511]

11.2. Risk factors for pregnancy-related venous thromboembolism

Pregnancy is accompanied by physiological changes leading to an increased VTE risk. First, increased procoagulant activity as well as decreased physiological anticoagulant and fibrinolytic activity result in a hypercoagulable state.[512,513] Second, the expanding uterus causes mechanical compression of the inferior vena cava and the pelvic veins, leading to impeded venous flow.[512,513] In addition, non-pregnancy-related and pregnancy- or delivery-related conditions may modify the individual VTE risk (see Supplementary data online, *Table S5*).

In pregnancy, as well as in the early post-partum period, an emerging suspicion of VTE requires immediate diagnostic clarification.

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11.3. Prevention of venous thromboembolism

Thromboprophylaxis follows an individualized risk assessment weighing the VTE risk against the bleeding risk ante- and post-partum.

11.3.1. Pregnant women with no prior indications for long-term anticoagulation

If prevention and treatment of VTE are initiated in the antepartum period, continuation for up to 6 weeks post-partum should be considered. In women with low-risk thrombophilia without a history of VTE, routine antepartum thromboprophylaxis is not required.[514] In women with a history of VTE or a high-risk thrombophilia, medical thromboprophylaxis is recommended (*Table 13*). In women with ovarian hyperstimulation syndrome after *in vitro* fertilization, thromboprophylaxis is recommended during the first trimester.[515,516]

The drug class of choice for the prevention and treatment of pregnancy-related VTE is LMWH.[517,518] Fondaparinux may be considered as an alternative. A randomized controlled trial comparing a weight-adjusted intermediate-dose LMWH with a fixed low-dose LMWH regimen found that weight adjustment did not reduce the risk of recurrent VTE in the combined ante- and post-partum periods.[519] Post-hoc analyses suggested a higher efficacy of weight-adjusted intermediate-dose LMWH in the post-partum period only, but this needs to be confirmed by future studies. In morbidly obese women, weight-based prophylactic dosing (considering anti-factor Xa measurement) instead of fixed dosing might be more appropriate.[198]

11.3.2. Pregnant women with prior indication for long-term anticoagulation

In case of pre-existing oral anticoagulation therapy due to previous VTE, oral anticoagulation (DOAC or VKA) should be replaced by LMWH at recognition of pregnancy.[517]

11.4. Management of acute venous thromboembolism

11.4.1. Clinical presentation and diagnosis

11.4.1.1. Deep vein thrombosis

The expanding uterus potentially reduces the blood flow in the iliocaval veins. In addition, a constitutional narrowing of the left-sided common iliac vein between the spine and the crossing artery could contribute to an increased risk of left-sided iliofemoral thrombosis.[520] The clinical LEFt criteria (L = Left, symptoms in the left leg; E = Edema, calf circumference difference ≥2 cm; Ft = First trimester of presentation) may be used to identify low risk of pregnancy-related DVT.[521,522] In pregnant women with suspicion of acute DVT, immediate diagnostic clarification is indicated (*Figure 9*).

Table 13 Reasons for antepartum/post-partum thromboprophylaxis

Medical conditions	Antepartum thromboprophylaxis	Post-partum thromboprophylaxis
History of unprovoked VTE	👍	👍
History of hormone-associated VTE	👍	👍
Homozygous factor V Leiden mutation	👍	👍
Heterozygous factor V Leiden mutation	👎	👎
Homozygous prothrombin gene mutation	👍[a]	👍
Heterozygous prothrombin gene mutation	👎	👎
Antithrombin deficiency	👍[a]	👍[a]
Antiphospholipid syndrome	👍[b]	👍
Protein C or S deficiency	👎	👍[a]
Combined thrombophilia	👍	👍

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VTE, venous thromboembolism.

👍 = yes; 👎 = no; 👍 = no clear evidence to administer or not—to be individualized.

[a]With family history of VTE (to be considered without family history of VTE).
[b]With history of VTE or pregnancy loss.

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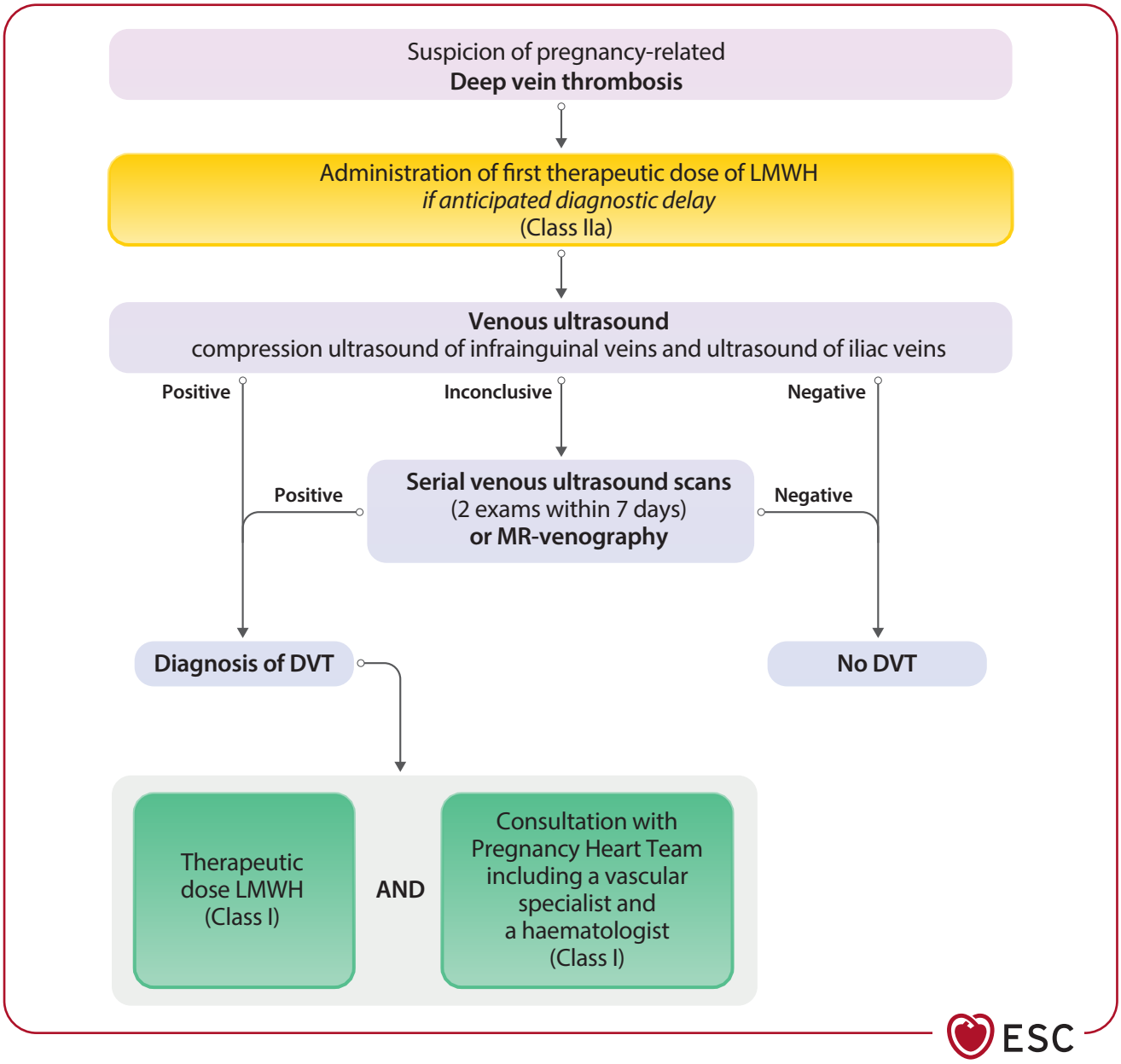

Figure 9 Algorithm for the diagnosis and treatment of deep vein thrombosis during pregnancy. DVT, deep vein thrombosis; LMWH, low-molecular-weight heparin, MR, magnetic resonance. Adapted from Chan *et al.*[523]

11.4.1.2. Pulmonary embolism

Clinical signs and symptoms of PE in pregnancy do not differ from those of PE in non-pregnant women. The diagnostic approach in haemodynamically stable pregnant women with suspicion of PE aims to reduce the need for computed tomography pulmonary angiography (CTPA) by implementing additional diagnostic strategies, such as clinical features, D-dimer, and venous ultrasound. Levels of D-dimer increase physiologically up to 39% per trimester.[524] A multinational study demonstrated the efficiency of a diagnostic strategy involving clinical probability, D-dimer measurements (threshold <500 µg/L), and peripheral venous compression ultrasound to reduce the need for CTPA.[525] Using both the YEARS criteria (1, clinical signs of acute DVT; 2, haemoptysis; 3, PE is the most likely diagnosis) and adapted D-dimer thresholds allows a further reduction in the need for CTPA (D-dimer threshold if YEARS criteria present <500 µg/L, if YEARS criteria absent <1000 µg/L).[526] Another meta-analysis confirmed the value of including D-dimer in a diagnostic algorithm to rule out PE in pregnant women with suspicion of PE (*Figure 10*).[527]

11.4.2. Treatment of venous thromboembolism in pregnancy

In pregnant women with a suspicion of VTE, anticoagulation with therapeutic LMWH should be commenced immediately, even before imaging, until the diagnosis of VTE is either excluded or confirmed. In pregnant women with confirmed acute VTE, therapeutic anticoagulation with weight-adjusted LMWH based on early pregnancy weight is recommended by using either a twice-daily or a once-daily regimen, each one resulting in a therapeutic daily dose[518] (see also *Section 5.2.1*). Currently, there is insufficient evidence to favour once- or twice-daily regimens.[217,218,518]

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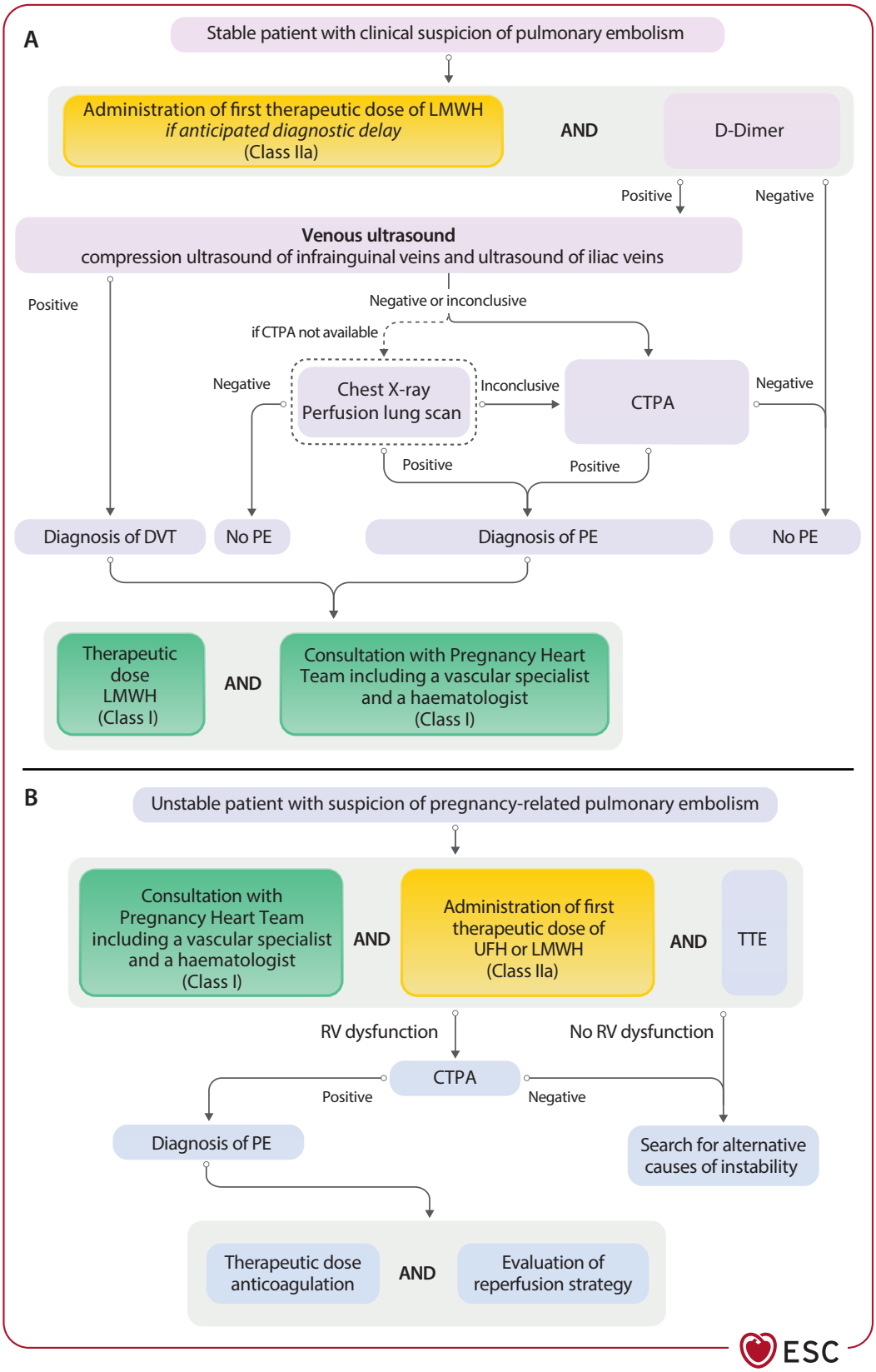

Figure 10 Algorithm for the diagnosis and treatment of pulmonary embolism in pregnancy in stable (A) and unstable women (B). CTPA, computed tomography pulmonary angiography; DVT, deep vein thrombosis; LMWH, low-molecular-weight heparin; PE, pulmonary embolism; RV, right ventricle; TTE, transthoracic echocardiogram; UFH, unfractionated heparin. Adapted from Barrios *et al.*[528] with permission from Elsevier.

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Despite fluctuations of anti-factor Xa levels during pregnancy, routine anti-factor Xa monitoring does not affect clinical outcomes and should only be considered in women with renal insufficiency or obesity.[211–213] Fondaparinux may be considered as an alternative. In unstable pregnant women with PE, UFH may be used in the initial phase of therapeutic anticoagulation.

After delivery, therapeutic-dose anticoagulation should be administered for a minimum of 6 weeks, up to an overall duration of 3 months, except for cases in which an indefinite duration of anticoagulation is indicated.[510,529] LMWH or VKAs may be given during lactation. A more detailed discussion of anticoagulants in the lactation period can be found in *Section 5*. Thrombolytic or interventional treatment of PE is not recommended in the peripartum period and should only be considered in women with high-risk PE after consultation with a specialized multidisciplinary team. In pregnant women with acute iliofemoral DVT, interventional thrombus removal should not routinely be performed. Data on the effectiveness and risks of the placement of temporary inferior vena cava filters for the prevention of PE in pregnant women are limited but appear to be comparable to non-pregnant women. Due to limited data and potential complications associated with the inferior vena cava filters, their placement should be limited to recurrent VTE despite appropriate anticoagulation or contraindication to therapeutic-dose anticoagulation therapy.[530,531]

11.5. Management of delivery and the post-partum period

For pregnant women receiving a prophylactic dose of anticoagulation, there is no specific need for a planned delivery. However, pregnant women receiving a therapeutic dose of anticoagulation need a planned delivery with prior discontinuation of LMWH to prevent spontaneous delivery in a period of full anticoagulation. Details for the management of anticoagulation during pregnancy and delivery, including for VTE, are provided in *Sections 4.5.6. and 5.2.1*.

12. Pregnancy in women with acquired heart disease

12.1. Acute chest pain in pregnancy

Diagnostic evaluation for chest pain in pregnant women follows the same protocol as in non-pregnant women, including clinical examination, ECG, biomarkers and echocardiography (*Figure 11*).[207,537,538] Importantly, spontaneous coronary artery dissection (SCAD) is a more prevalent cause of chest pain during pregnancy and in the early post-partum period than in non-pregnant women.[539]

Treatment and management of the specific differential diagnoses should follow established respective guidelines. The specificity of D-dimer is reduced during pregnancy[540,541] and women should not undergo chest CT based solely on D-dimer levels (*Section 11*). In suspected acute aortic syndromes, there should be a low threshold for aortic CT and consultation with the aortic team in emergencies (*Section 8*).[542]

12.2. Coronary artery disease

12.2.1. Acute coronary syndrome

12.2.1.1. Coronary artery disease epidemiology and aetiology

Acute coronary syndromes (ACS) are a major cause of maternal death in developed countries, accounting for 20% of cardiovascular deaths.[543] The risk of ACS is three to four times higher in pregnant women than in non-pregnant women of reproductive age,[544] and mortality is estimated at 5%.[545] Because the age at giving birth is increasing overall, ACS in pregnancy may become more common.[546] Although ACS can occur at any stage of pregnancy, it is more common in the third trimester or post-partum.[544] Classic ASCVD risk factors are associated with ACS during pregnancy.

Recommendation Table 11 — Recommendations for venous thromboembolic diseases and pregnancy (see Evidence Tables 9 and 10)

Recommendations	Class[a]	Level[b]
For pregnant or post-partum women at high risk[c] of VTE, a prophylactic fixed dose of LMWH is recommended over a higher weight-adjusted dose to reduce the risk of VTE.[519]	I	B
In pregnant women or women in the post-partum period with suspicion of VTE (DVT and/or PE), an immediate formal diagnostic assessment with validated methods is recommended and should not be postponed.[525,526]	I	B
In pregnant women or women in the post-partum period with newly diagnosed VTE (DVT and/or PE), the involvement of the Pregnancy Heart Team, including a vascular specialist and a haematologist, is recommended.	I	C
In pregnant or post-partum women with a diagnosis of VTE without haemodynamic instability, anticoagulation is recommended by using therapeutic-dose LMWH based on early pregnancy body weight.[212,532]	I	C
In pregnant women or women in the post-partum period with a strong clinical suspicion of VTE, initiation of treatment with a therapeutic dose of LMWH should be considered until the presence of VTE has been ruled out or confirmed.	IIa	C
In pregnant or post-partum women with a diagnosis of acute high-risk PE,[d] a catheter-based reperfusion strategy or systemic thrombolysis should be considered.[533–535]	IIa	C
In pregnant or post-partum women with a diagnosis of acute high-risk PE,[d] surgical thrombectomy may be considered as an alternative to a catheter-based approach or systemic thrombolysis.[533–535]	IIb	C

DVT, deep vein thrombosis; LMWH, low-molecular-weight heparin; PE, pulmonary embolism; VTE, venous thromboembolism.
[a]Class of recommendation.
[b]Level of evidence.
[c]See Supplementary data online, *Table S5* for VTE risk factors.
[d]According to the Pulmonary Embolism Severity Index from the 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism.[536]

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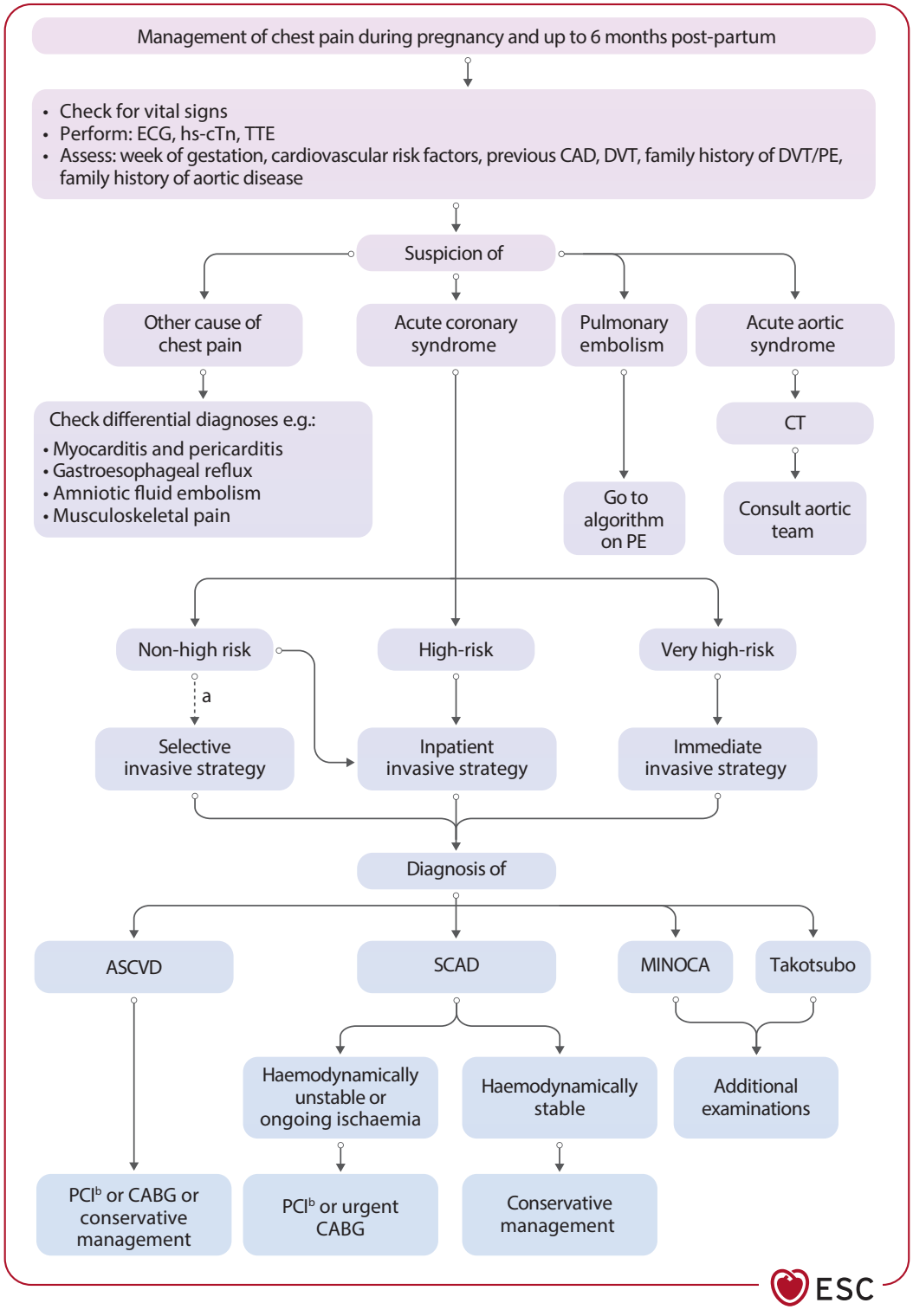

Figure 11 Management of chest pain during pregnancy and within 6 months post-partum. ASCVD, atherosclerotic cardiovascular disease; B-blocker, beta-blocker; CABG, coronary artery bypass graft; CAD, coronary artery disease; CT, computed tomography; DVT, deep venous thrombosis; ECG, electrocardiogram; EF, ejection fraction; hs-cTn, high sensitivity cardiac troponin; i.v., intravenous; MINOCA, myocardial infarction with non-obstructive coronary arteries; o.d., once a day; PCI, percutaneous coronary intervention; PE; pulmonary embolism; SCAD, spontaneous coronary artery dissection; TTE, transthoracic echocardiogram. [a]In patients without very high or high-risk features and a low index of suspicion for unstable angina. [b]Dual anti-platelet therapy: clopidogrel: loading dose of 300–600 mg orally, followed by an oral maintenance dose of 75 mg o.d. Aspirin: loading dose of 150–300 mg orally or 75–250 mg i.v. if oral ingestion is not possible, followed by an oral maintenance dose of 75–100 mg o.d.

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Additional risk factors are pre-eclampsia, thrombophilia, transfusion, post-partum infection, multiparity, and PPH. Pregnancies that required fertility treatment have not been shown to have an increased risk of ACS.[547]

Pregnancy-associated SCAD is the single most frequent cause of ACS during pregnancy and post-partum (43%),[548] followed by atherosclerotic lesion (27%), coronary embolism (17%), and vasospasm (2%).[239] Coronary thromboembolism may be due to pregnancy-related hypercoagulability and paradoxical embolization.[549] Vasospasm has been associated with the use of ergot derivatives prescribed for lactation suppression or to treat PPH.[549]

12.2.1.2. Presentation and diagnosis

Clinical presentation of ACS in pregnancy is the same as in the non-pregnant population. However, pregnant women with SCAD tend to have a more severe clinical presentation than women with non-SCAD ACS.[550–553]

An ACS in pregnancy should be suspected in women presenting with cardiac arrest, acute onset chest pain, dyspnoea, ischaemic changes on ECG, or elevated cardiac biomarkers.[551,554] Diagnostic evaluation should follow ACS strategies (*Figure 11*).[555–557] Major ischaemic ECG changes due to pregnancy itself are not expected (*Figure 1*).

Invasive coronary angiography during pregnancy should be reserved for those with ACS, or when other diagnostic methods are inappropriate. ECG changes, such as transient ST-segment depression and T-wave inversion can be normal during pregnancy, but a serum troponin rise suggests myocardial injury as in non-pregnant women. ST elevation is not normal in pregnancy and warrants urgent attention.[558] ST-elevation myocardial infarction (STEMI) in pregnant women involves the anterior wall in 70%–80% of cases.[239,558] In more than half the cases, a reduction of LVEF <40% is observed, leading to a high incidence of complications.[20] Myocardial infarction with non-obstructive coronary arteries (MINOCA) should be considered a working diagnosis warranting further investigation.[559]

12.2.1.3. Pregnancy-associated spontaneous coronary artery dissection

Pregnancy-associated SCAD affects 1.81 per 100 000 pregnancies[560] and may occur at any time during or after pregnancy, although >70% occur early post-partum, most commonly within the first week.[550] Multiple predisposing factors have been described, including oestrogen and progesterone surges causing structural changes to the coronary tunica media. SCAD predominantly occurs in the left-sided coronaries, with multivessel involvement.[561]

Percutaneous coronary intervention (PCI) in SCAD is associated with an increased risk of complications, particularly iatrogenic dissection and haematoma extension.[562] For this reason, a conservative approach to revascularization is advised in clinically stable women with SCAD without active or ongoing ischaemia.[539,563] If SCAD involves the left main coronary artery or proximal vessels, a coronary artery bypass graft (CABG) may be considered depending on technical considerations and local expertise.[539] A multidisciplinary team should decide whether the patient is a candidate for PCI or CABG.

Optimal medical management following SCAD is unknown but is currently being investigated in an ongoing clinical trial.[562] Limited observational data suggest that beta-blockers (e.g. labetalol) and avoiding hypertension may be associated with a lower risk of recurrent SCAD.[564,565] The role of antiplatelet therapies in conservatively managed SCAD has been controversial, with evidence favouring single antiplatelet therapy with aspirin.[563,566] Women with a history of SCAD should be carefully counselled regarding the risk of recurrent events in subsequent pregnancy.[567,568]

12.2.2. Coronary artery interventions

The indications for acute revascularization are comparable to those for non-pregnant women. In patients with high or very high-risk ACS, immediate coronary angiography and PCI, if indicated, are recommended.[557] Moreover, an early invasive strategy with coronary angiography is recommended for pregnant women with a confirmed or a working diagnosis of non-ST-elevation ACS (NSTE ACS) and with a high index of suspicion for unstable angina. In atherosclerotic lesions[557] PCI is indicated when there is ongoing or recurrent chest pain, haemodynamic instability, or ongoing ischaemia due to functionally significant coronary stenoses or acute occlusions.[122]

The choice of coronary stents should not be different from that for non-pregnant women. Duration of DAPT should follow recommendations for non-pregnant patients with an individual approach considering ischaemic and delivery-related risks, including bleeding risk during delivery and neuraxial anaesthesia. Stents approved for short DAPT may be preferred during pregnancy in specific cases according to gestational age and timing of delivery. In the case of coronary embolism, thrombo-aspiration and/or a simple angioplasty can be performed.[43,569] Invasive procedures should follow the ALARA principle (see *Section 4.3.5*).

Systemic thrombolysis may be an alternative reperfusion strategy if timely PCI is not available. Recombinant tissue plasminogen activator does not cross the placenta but can induce bleeding complications, including subplacental.

12.2.3. Chronic coronary syndromes in pregnancy

Pregnant women with chronic coronary syndromes (CCS) are at high risk of adverse maternal and foetal outcomes: 32% have cardiovascular complications (including 9% with ischaemic cardiovascular complications) and there is 2% maternal mortality.[570] The CARPREG II score, now also included in the mWHO 2.0 classification (*Table 6*, *Section 4*), highlights the high risk of CCS as a predictor of maternal complications. When counselling women with CCS, pregnancy can preferably be considered when there is no residual ischaemia or LV dysfunction 12 months after an index event.

12.2.4. Management and delivery

Women with ACS or CCS should be managed by a Pregnancy Heart Team. Treatment should be tailored to the underlying pathophysiology, although foetal considerations may affect the choice of therapy.[50,571] All pregnant women with ACS and their foetus should be monitored at an intensive cardiac care unit.

The mode of delivery in a patient with gestational ACS or CCS should be determined by obstetric considerations and the clinical status of the mother. A vaginal delivery is indicated in most women with obstructive coronary artery disease (CAD). Vaginal delivery eliminates the potential risks associated with general anaesthesia and a major surgical procedure. Clopidogrel must be withheld a minimum of 5 days before neuraxial anaesthesia to reduce the risk of epidural hematoma.[572] An elective caesarean section avoids a long or stressful labour and allows better control of the time of delivery. A plan for emergency delivery of a potentially viable foetus in case of sudden maternal deterioration should also be established. An arbitrary minimum time to delivery in stable women is at 2 weeks after ACS.[18]

Lipid-lowering and antiplatelet therapy are described in *Section 5*.

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Recommendation Table 12 — Recommendations for coronary artery disease and pregnancy (see Evidence Table 11)

Recommendations	Class[a]	Level[b]
In pregnant women with chest pain, it is recommended to exclude life-threatening cardiovascular conditions, including PE, ACS (including SCAD), and acute aortic syndrome.[538,539]	I	C
It is recommended to manage pregnant women with ACS in the same way as non-pregnant women, including diagnostic investigations and interventions.[557]	I	C
Low-dose ASA is recommended as the antiplatelet treatment of choice during pregnancy and lactation when single antiplatelet treatment is indicated.[573–579]	I	B
If DAPT is required, clopidogrel is recommended as the P2Y12 inhibitor of choice during pregnancy.[239]	I	C
The duration of DAPT (aspirin and clopidogrel) in pregnant women undergoing coronary stent implantation is recommended to be the same as in non-pregnant women, with an individual approach considering ischaemic risk and delivery-related bleeding risks.[239]	I	C
A vaginal delivery should be considered in most pregnant women with ACS, depending on LV function and clinical symptoms.	IIa	C
Continuation of statins may be considered during pregnancy in women with established ASCVD.[580–582]	IIb	C

ACS, acute coronary syndrome; ASA, acetylsalicylic acid; ASCVD, atherosclerotic cardiovascular disease; DAPT, dual antiplatelet therapy; LV, left ventricular; PE, pulmonary embolism; SCAD, spontaneous coronary artery dissection.
[a]Class of recommendation.
[b]Level of evidence.

12.3. Hypertensive disorders

Hypertensive disorders of pregnancy are the second most common medical complications (after PPH), affecting 5%–15% of pregnancies worldwide, and are a major cause of maternal, foetal, and neonatal morbidity and mortality.[583] Hypertensive disorders in pregnancy include pre-existing hypertension (chronic hypertension), gestational hypertension, and pre-eclampsia (*Table 14*). Over recent years an upward incidence trend has been observed due to older age at first childbirth and rising prevalence of obesity.[586,587] Maternal risks include placental abruption, stroke, multiple organ failure, and disseminated intravascular coagulation.[588]

Table 14 Hypertensive disorders of pregnancy

A. Pre-existing (chronic) hypertension
Hypertension which either precedes pregnancy or develops before 20 weeks gestation, usually persisting >6 weeks post-partum, and which may be associated with proteinuria. (1) Primary hypertension (2) Secondary hypertension (3) White-coat hypertension (4) Masked hypertension
B. Gestational hypertension
Hypertension which develops after 20 weeks gestation and usually resolves within 6 weeks post-partum. **Transient gestational hypertension** Usually detected in the clinic but then settles with repeated BP measurements taken over several hours; associated with a 40% risk of developing true gestational hypertension or pre-eclampsia in the remainder of the pregnancy, thus requiring careful follow-up.
C. Pre-eclampsia

Gestational hypertension accompanied by one or more of the following new-onset conditions at or after 20 weeks gestation:
• Proteinuria [urinary albumin excretion in a 24 h urine sample >0.3 g/day or UACR in a random spot urine sample >30 mg/mmol (0.3 mg/mg)]
• Other maternal organ dysfunction including:
  • Acute kidney injury (serum creatinine ≥90 μmol/L; 1 mg/dL)
  • Liver dysfunction (elevated ALT or AST >40 IU/L; >0.67 μkat/L with or without right upper quadrant or epigastric abdominal pain)
  • Neurological complications (e.g. eclampsia/convulsions, altered mental status, blindness, stroke, clonus, severe headaches, persistent visual scotomata)
  • Haematological complications (platelet count <150 000/μL, disseminated intravascular coagulation, haemolysis)
• Uteroplacental dysfunction (IUGR, abnormal umbilical artery Doppler waveform analysis, or stillbirth).

D. Pre-existing hypertension + superimposed pre-eclampsia
Pre-existing hypertension associated with any of the above maternal organ dysfunctions consistent with pre-eclampsia or a further increase in BP with new-onset proteinuria.
E. Antenatally unclassifiable hypertension
When BP is first recorded after 20 weeks gestation and hypertension is diagnosed, reassessment is necessary at or after 6 weeks post-partum. If hypertension resolves, it should be reclassified as gestational hypertension, whereas if hypertension persists, it should be reclassified as pre-existing/chronic hypertension.

Based on Mancia *et al.*[584] and McEvoy *et al.*[585]
ALT, alanine transaminase; AST, aspartate aminotransferase; BP, blood pressure; IUGR, intrauterine growth restriction; UACR, urine albumin–creatinine ratio.

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The foetus is at high risk of IUGR prematurity, and intrauterine death (25%, 27%, and 4%, respectively, in cases of pre-eclampsia).[589]

12.3.1. Definition and classification of hypertension in pregnancy

Hypertension in pregnancy is typically defined as systolic blood pressure (BP) of ≥140 mmHg and/or diastolic BP of ≥90 mmHg,[585] measured using repeated BP readings in the office or hospital on two separate occasions or ≥15 min apart in severe hypertension (≥160/110 mmHg).[584,590]

12.3.1.1. Pre-eclampsia/eclampsia

Pre-eclampsia is defined as gestational hypertension complicated by new onset of the laboratory or clinical changes reported in *Table 14* at or after 20 weeks. Eclampsia is defined as the new onset of seizures or coma in a pregnant woman with pre-eclampsia.[584,585]

The combination of haemolysis, thrombocytopenia, and elevated transaminases defines HELLP syndrome, which is usually considered to be a variant of pre-eclampsia.[43] Risk factors for pre-eclampsia are described in *Table 15*.

Table 15 Risk factors for pre-eclampsia

High risk factors for pre-eclampsia
Hypertensive disorders during a previous pregnancy
Chronic hypertension
Chronic kidney disease
Type 1 or type 2 diabetes mellitus
Autoimmune diseases such as systemic lupus erythematosus or antiphospholipid syndrome
Assisted reproductive therapy in the current pregnancy
Moderate risk factors for pre-eclampsia
Nulliparity
Age ≥40 years
Pregnancy interval of more than 10 years
BMI ≥35 kg/m^2 at the first visit
Family history of pre-eclampsia
Multi-foetal pregnancy

Based on Mancia *et al.*[584] and McEvoy *et al.*[585]
BMI, body mass index.

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12.3.2. Diagnosis and risk assessment

12.3.2.1. Blood pressure measurement

Maternal BP should be assessed at each encounter and should be measured in the sitting position (or the left lateral recumbent position during labour) with an appropriately sized arm cuff at heart level and using Korotkoff V for diastolic BP. Mercury sphygmomanometers remain the gold standard for BP measurement in pregnancy as automatic devices tend to under-record BP and are unreliable in severe pre-eclampsia. Only automatic devices specifically validated for pregnancy should be used.[584,591]

The diagnosis of hypertension in pregnancy by ambulatory BP monitoring is superior to office BP measurements or home BP monitoring for the prediction of pregnancy outcomes.[584,592] Ambulatory BP monitoring avoids unnecessary treatment of white-coat hypertension, and is useful in the management of high-risk pregnant women with hypertension and those with diabetic or hypertensive nephropathy.[593,594] Either home BP monitoring or office BP measurements may be used alternatively or complementarily to diagnose hypertensive disorders during pregnancy in women at risk of pre-eclampsia.

However, among pregnant women with pre-existing or gestational hypertension, home BP monitoring is not associated with better BP control compared with scheduled office BP measurements.[584,595,596] Either can be used for BP monitoring.[595,596]

12.3.2.2. Laboratory tests

Basic laboratory investigations recommended for monitoring hypertensive disorders of pregnancy include urinalysis, blood count, haematocrit, liver enzymes, serum creatinine, and serum uric acid. All pregnant women should be assessed for proteinuria in early pregnancy to detect pre-existing renal disease and, in the second half of pregnancy, to screen for pre-eclampsia.[43]

In addition to basic laboratory tests, the following investigations may be considered:

- Ultrasound of the adrenals, and plasma and urinary fractionated metanephrine assays.
- Doppler ultrasound of uterine arteries (after 20 weeks of gestation).[597]
- A ratio of soluble fms-like tyrosine kinase-1 (sFlt-1) to placental growth factor (PlGF) of <38 can be used to reliably exclude the development of pre-eclampsia over the next 7 days when clinically suspected.[43,598]

If women with chronic hypertension are suspected of developing pre-eclampsia, testing for PlGF may help rule out pre-eclampsia between 20 and 36 weeks.[599,600]

12.3.3. Management of hypertension in pregnancy

Management of hypertension in pregnancy depends on the woman's BP and gestational age, and the presence of associated maternal and foetal risk factors.[601,602] In pregnant women with BP >160/110 mmHg, hospital admission is recommended[603] (*Figure 12A*). If BP is ≥140–159/90–99 mmHg, BP control is mandatory with a BP target of <140/90 mmHg.

In two large trials,[583,604] tight diastolic BP control (<85–90 mmHg) in women with pre-existing hypertension was superior to less tight diastolic BP control (<100–105 mmHg) and caused no harm.

In pregnant women with diagnosed hypertension, blood tests, clinical examination, and assessment of proteinuria are mandatory and should be repeated regularly.[599] If a urine dipstick is positive or borderline for proteinuria, and symptoms and laboratory tests (including biomarker assessment) are indicative of pre-eclampsia, the diagnosis of pre-eclampsia can be made and hospital admission is recommended where there are clinical concerns.[599]

12.3.3.1. Non-pharmacological management

Pregnant women with hypertension should be advised to follow a healthy lifestyle including physical activity, smoking cessation, a healthy diet, and control of body weight. Lifestyle changes before and during pregnancy may ameliorate both maternal and foetal risks.[605] Salt restriction is not advised to reduce hypertensive disorders during pregnancy, but it is reasonable for women with pre-existing hypertension to continue with a low sodium diet.[584] Unless contraindicated, aerobic exercise should be recommended in pregnant women with hypertension to maintain ideal body weight and reduce adverse pregnancy

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outcomes.[606] Obese women are advised to avoid an increase in weight of more than ~7 kg from pre-conception.[601]

Calcium supplements are recommended for the prevention of pre-eclampsia in women with a low dietary intake of calcium (<600 mg/day),[607] where low-dose calcium supplementation (<1 g/day) has been shown to be as effective as high-dose (≥1 g/day).[608]

12.3.3.2. Pharmacological treatment

12.3.3.2.1. Mild hypertension (BP 140/90 – 159/109 mmHg). In mild gestational hypertension it seems reasonable to initiate treatment at BP values of 140/90 mmHg,[583,609] whereas a diastolic BP reduction to <80 mmHg is not recommended.

Methyldopa, beta-blockers (most data are available for labetalol), and dihydropyridine CCBs (most data are available for nifedipine) are the drugs of choice for mild gestational and pre-existing hypertension.[584]

ACE-Is, ARBs, and direct renin inhibitors are strictly contraindicated. Diuretics are not advised in gestational hypertension and pre-eclampsia, due to the reduction of intravascular volume and reduction of uteroplacental perfusion and thereby possible foetal adverse effects.

12.3.3.2.2. Pre-eclampsia. Pre-eclampsia may require hospital admission, but not all women will require ongoing hospitalization and care should be individualized (*Figure 12B and Figure 12C*). Women with at least one high-risk factor or two moderate-risk factors for pre-eclampsia (*Table 15*) should be advised to take 75–150 mg aspirin daily at bedtime from week 12 to week 36/37.[599,610] We advise discontinuation of low-dose aspirin at week 36/37 when the aspirin indication is pre-eclampsia.

Pre-eclampsia with severe features (severe hypertension with or without proteinuria, any hypertension grade with neurological, haematological, or cardiovascular complications, liver dysfunction, or renal dysfunction) should be managed with a magnesium sulfate infusion to prevent eclampsia, in addition to early delivery.[43,611] Magnesium toxicity can present with cardiac effects, including ECG interval changes (prolonged PR, QRS, and QT intervals) at magnesium levels of 2.5–5 mmol/L, and can progress to atrioventricular (AV) nodal conduction block, bradycardia, hypotension, and cardiac arrest at levels of 6–10 mmol/L. If magnesium toxicity is suspected, it is recommended to stop the magnesium infusion and give 30 mL i.v. calcium gluconate.[612–614]

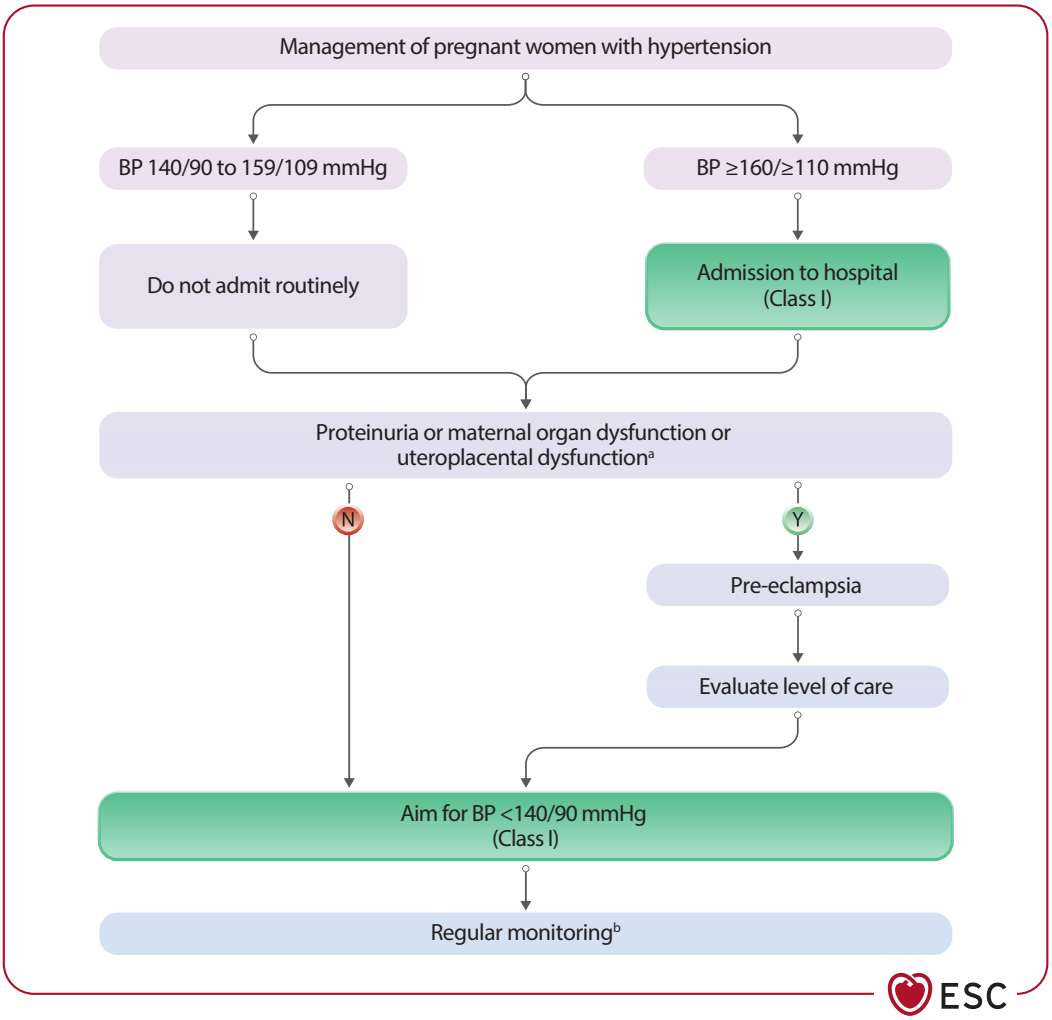

Figure 12A Management of hypertension and pre-eclampsia in the emergency ward. BP, blood pressure; N, no; Y, yes.
[a] See Figure 12B.
[b] See Figure 12C.

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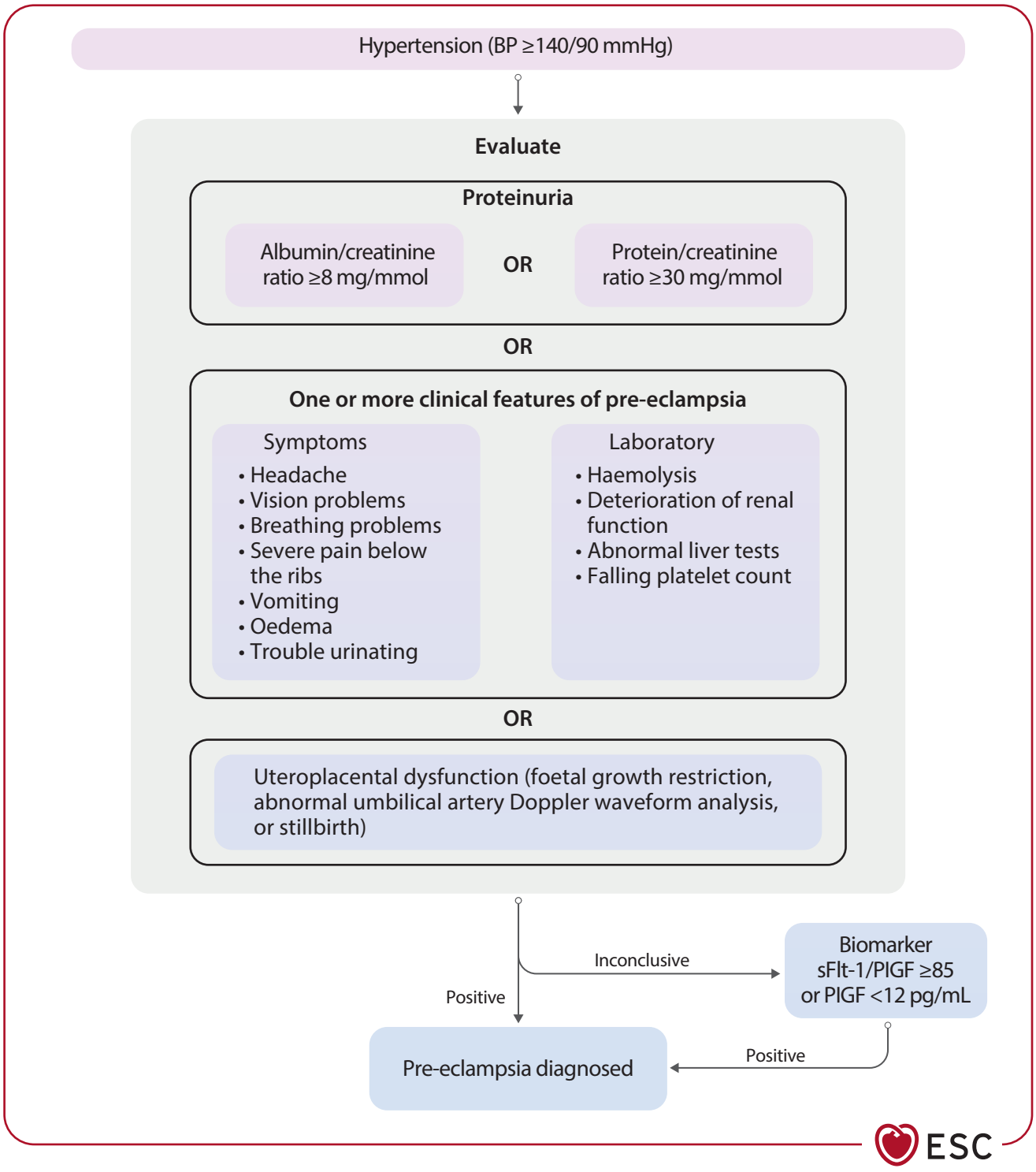

Figure 12B Proteinuria assessment and diagnosis of pre-eclampsia. BP, blood pressure; PlGF, placental growth factor; sFLt-1, soluble fms-like tyrosine kinase-1.

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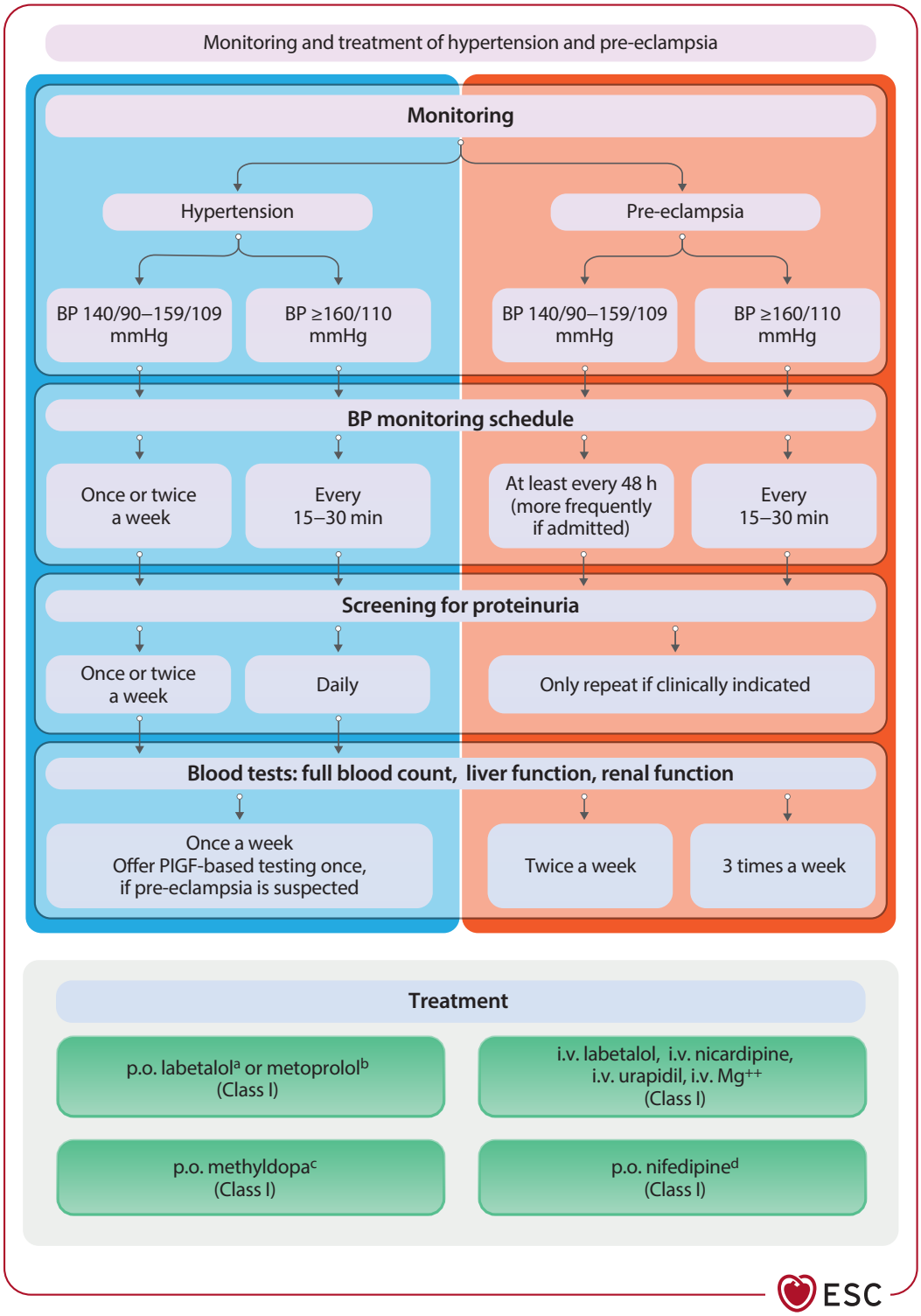
Monitoring and treatment of hypertension and pre-eclampsia
Monitoring
Hypertension
Pre-eclampsia
BP 140/90–159/109 mmHg
BP ≥160/110 mmHg
BP 140/90–159/109 mmHg
BP ≥160/110 mmHg
BP monitoring schedule
Once or twice a week
Every 15–30 min
At least every 48 h (more frequently if admitted)
Every 15–30 min
Screening for proteinuria
Once or twice a week
Daily
Only repeat if clinically indicated
Blood tests: full blood count, liver function, renal function
Once a week
Offer PIGF-based testing once, if pre-eclampsia is suspected
Twice a week
3 times a week
Treatment
p.o. labetalol[a] or metoprolol[b] (Class I)
i.v. labetalol, i.v. nicardipine, i.v. urapidil, i.v. Mg++ (Class I)
p.o. methyldopa[c] (Class I)
p.o. nifedipine[d] (Class I)
ESC

Figure 12C Monitoring and treatment of hypertension and pre-eclampsia. Mg, magnesium; PlGF, placental growth factor; p.o., per oral.
[a]Labetalol 100 mg p.o. b.i.d. [b]Metoprolol 100 mg p.o. b.i.d. [c]Methyldopa 250 mg p.o. b.i.d./t.i.d. [d]Nifedipine 5–10 mg p.o. 10 mg p.o. if >160/110 mmHg.

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12.3.3.2.3. Severe hypertension (BP ≥160/110 mmHg). In severe hypertension, assessment and gradual BP reduction to <160/110 mmHg is mandatory in a hospital setting. Continuous cardiotocographic monitoring is also compulsory. The selection of antihypertensive drugs and the route of administration depend on initial diagnosis, expected delivery time, presence or absence of pre-eclampsia, and the preferences and experience of the attending physicians.[43,611] Recent comprehensive meta-analyses comparing commonly used antihypertensive drugs (e.g. oral nifedipine, labetalol, methyldopa) (Figure 12C) showed they had similar efficacy in lowering BP in severe hypertension in pregnancy, with nifedipine showing superiority in some studies (see *Section 5.2.6*).[43,253,254,611,615] Nifedipine also has the advantage of wider distribution, availability, and lower pricing. Available data showed that hydralazine is less effective than other drugs and is associated with more side effects.[616] Hydralazine is therefore a second-line option to be used only if other drugs are not available.

In cases of pre-eclampsia with severe features, persistent severe hypertension, or recurrent severe hypertension despite orally administered agents, i.v. administration of labetalol (or nicardipine) is advised. Intravenous urapidil can also be used but may not be available in all countries.[611,617,618] Sodium nitroprusside is the drug of last resort because prolonged treatment is associated with an increased risk of foetal cyanide poisoning.[619] The drug of choice when pre-eclampsia is associated with pulmonary oedema is nitroglycerine (glyceryl trinitrate), given as an i.v. infusion of 5 µg/min, gradually increasing every 3–5 min to a maximum dose of 100 µg/min, in combination with diuretics.[43,611]

12.3.4. Hypertension and delivery

A recent randomized controlled trial from the United Kingdom found that once a diagnosis of pre-eclampsia was made in women with late pre-term pre-eclampsia (34–37 weeks), there was a lower rate of maternal morbidity and less severe maternal hypertension when delivery was planned to occur within the next 48 h.[620] However, a greater proportion of neonates were admitted to the neonatal intensive care unit compared to mothers who were managed from an earlier stage. Decisions around timing of delivery should be individualized considering both maternal and foetal well-being. If well managed hypertension alone, delivery should be planned around 39 weeks of gestation.

When choosing the mode of delivery, the clinical scenario should take into consideration the current gestational age and a full foetal assessment, as well as the preference of the mother. If induction of labour is to be considered, continuous foetal monitoring is required. Women with severe pre-eclampsia benefit from neuraxial anaesthesia to reduce the hypertensive response to pain. Additionally, an epidural will provide adequate anaesthesia should a caesarean delivery be required.

After delivery, methyldopa should be avoided due to the risk of post-partum depression. Methyldopa should be stopped within 2 days after delivery and changed to an alternative treatment.

Recommendation Table 13 — Recommendations for hypertensive disorders and pregnancy (see Evidence Tables 12–17)

Recommendations	Class[a]	Level[b]
It is recommended to aim for systolic BP <140 mmHg and diastolic BP <90 mmHg in pregnant women.[583,585,609]	I	B
Systolic BP ≥160 mmHg or diastolic BP ≥110 mmHg in a pregnant woman is an emergency, and treatment in a hospital setting is recommended.[43,584]	I	C
Low-dose aspirin (75–150 mg daily) is recommended in women at moderate or high risk of pre-eclampsia (i.e. at least one high-risk factor or two moderate-risk factors for pre-eclampsia) from weeks 12 to 36/37.[610,621,622]	I	A
In women with gestational hypertension, initiation of drug treatment is recommended at systolic BP ≥140 mmHg or diastolic BP ≥90 mmHg.[c583–585,609]	I	B
Methyldopa is recommended for the treatment of hypertension in pregnancy.[623,624]	I	B
Labetalol, metoprolol, and dihydropyridine CCBs are recommended for the treatment of hypertension in pregnancy.[623]	I	C
In severe hypertension, drug treatment with i.v. labetalol, urapidil, nicardipine, or oral short acting nifedipine or methyldopa is recommended for acute reduction in blood pressure.[625,626] Intravenous hydralazine is a second-line option.	I	C
In pre-eclampsia associated with pulmonary oedema, nitroglycerine given as an i.v. infusion is recommended.[627]	I	C
In women with pre-eclampsia without severe features, delivery is recommended at 37 weeks.[620,628]	I	B
It is recommended to expedite delivery in women with pre-eclampsia associated with adverse markers such as haemostatic disorders.[629]	I	C
In women with gestational hypertension, delivery is recommended at 39 weeks.[620,628]	I	B
Ambulatory BP or home BP monitoring should be considered to exclude white-coat and masked hypertension, which are common in pregnancy.	IIa	C
Home BP monitoring may be considered as an adjunct to office BP measurements in pregnant women to detect new-onset hypertension or for monitoring BP control.[595,596]	IIb	B

BP, blood pressure; CCB, calcium channel blocker; i.v, intravenous.
[a]Class of recommendation.
[b]Level of evidence.
[c]Office BP measurements.

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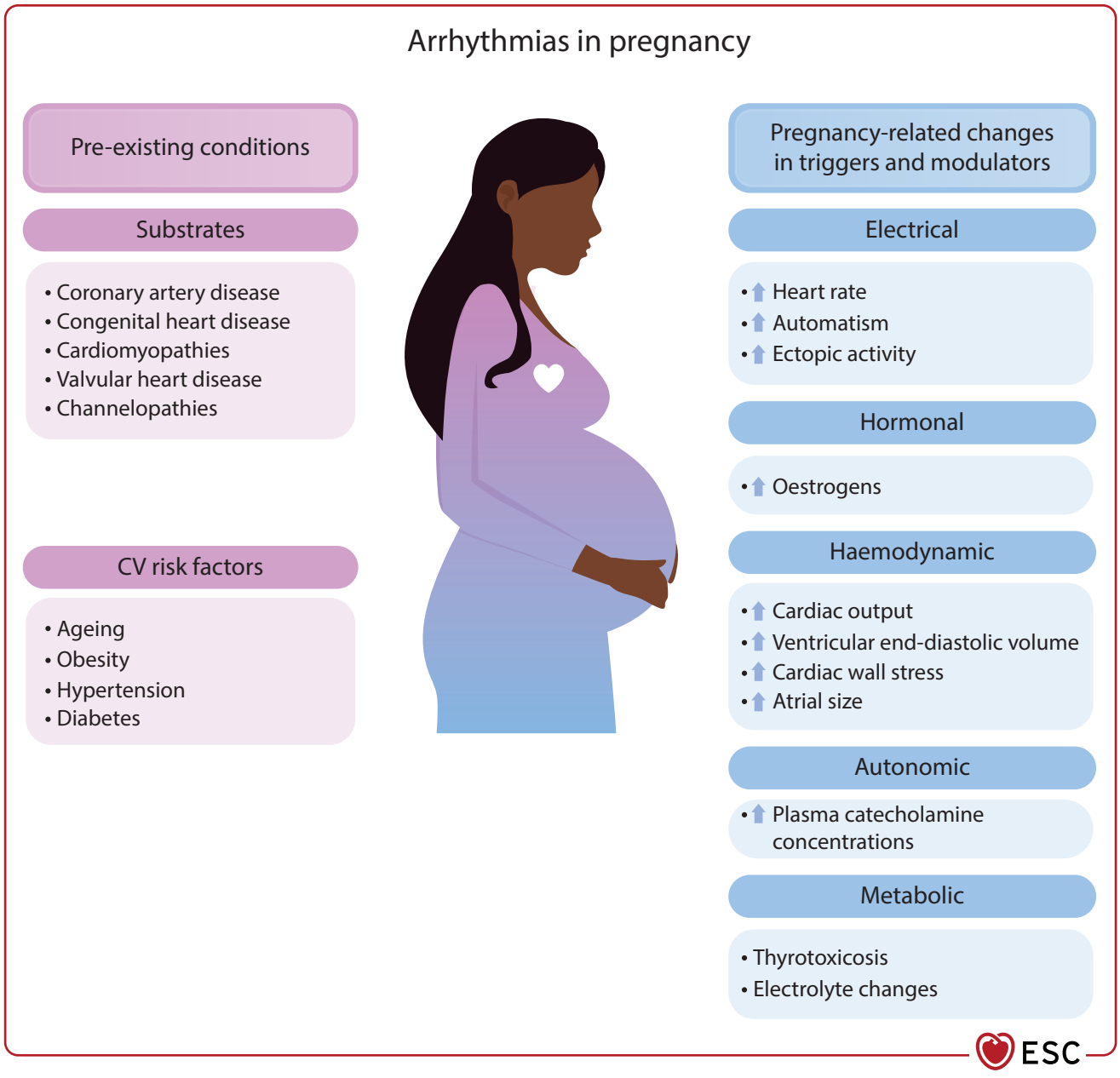

Figure 13 Arrhythmogenesis in pregnant women. CV; cardiovascular.

12.4. Arrhythmias

The number of pregnant women presenting with arrhythmia is rising due to increasing age at pregnancy and cardiovascular risk factors such as obesity, hypertension, diabetes, and CAD in pregnant women.[630] Haemodynamic, metabolic, and hormonal changes, and changes in autonomic function may contribute to increased arrhythmogenesis during pregnancy and women with prior history of arrhythmia may experience worsening of symptoms during pregnancy (*Figure 13*).[630] Arrhythmia may have serious effects on the health of both the mother and the foetus and should be treated similarly to arrhythmias in non-pregnant women.

12.4.1. Supraventricular arrhythmias

12.4.1.1. New-onset narrow QRS tachycardia

New-onset narrow QRS (<120 ms) tachycardias in pregnancy are treated according to haemodynamic stability. In all cases with haemodynamic instability caused by any supraventricular tachycardia (SVT) including AF and AFL, synchronized, direct current cardioversion is indicated (*Figure 14*).[631] The foetal heart rate should be closely monitored after cardioversion.[632] In haemodynamically stable narrow QRS tachycardias, the use of vagal manoeuvres (modified Valsalva manoeuvre, carotid sinus massage) may terminate an atrioventricular (nodal) re-entry tachycardia (AV(N)RT) arrhythmia.[633]

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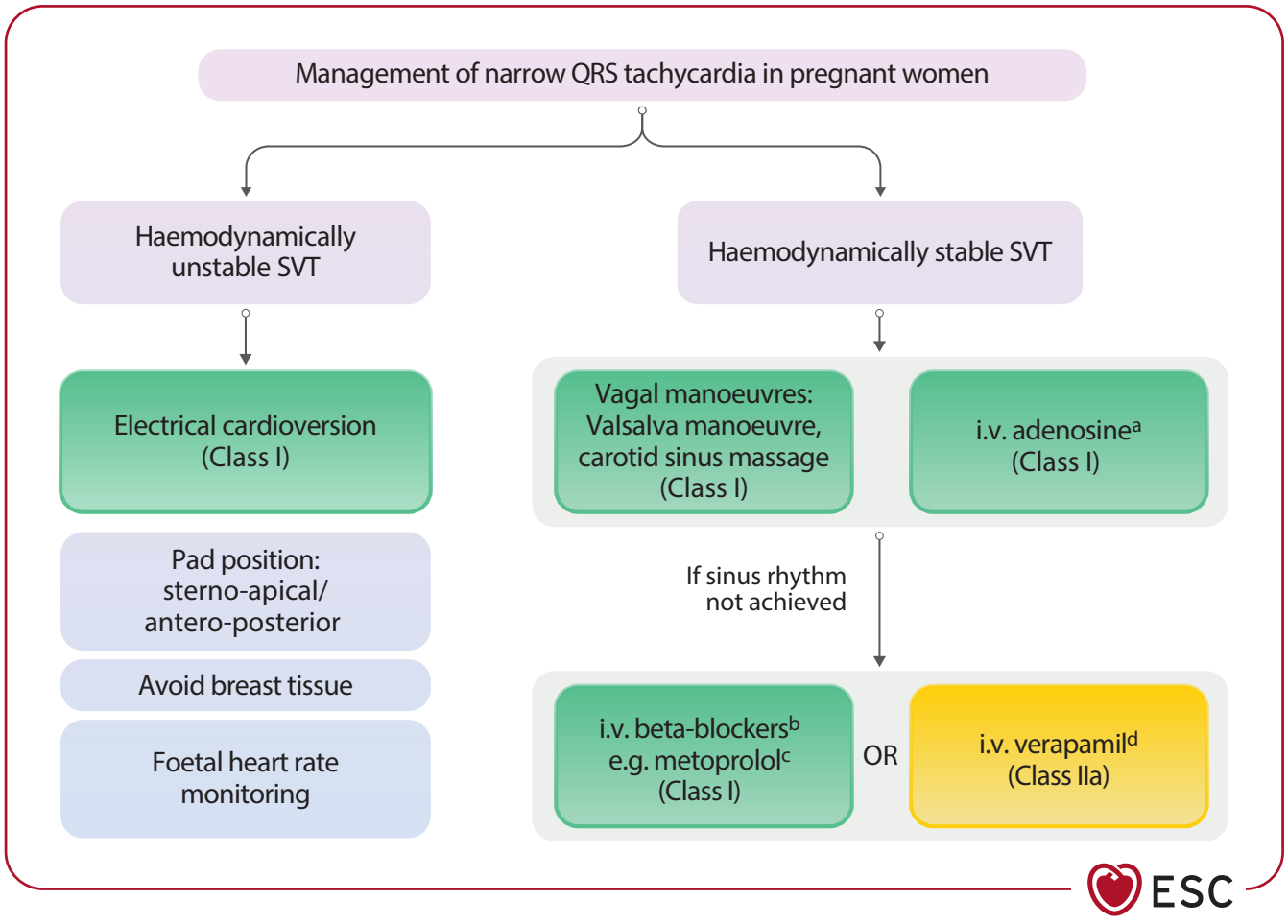
Management of narrow QRS tachycardia in pregnant women
Haemodynamically unstable SVT
Haemodynamically stable SVT
Electrical cardioversion (Class I)
Vagal manoeuvres: Valsalva manoeuvre, carotid sinus massage (Class I)
i.v. adenosine[a] (Class I)
Pad position: sterno-apical/ antero-posterior
Avoid breast tissue
Foetal heart rate monitoring
If sinus rhythm not achieved
i.v. beta-blockers[b] e.g. metoprolol[c] (Class I)
OR
i.v. verapamil[d] (Class IIa)
ESC

Figure 14 Management of narrow QRS tachycardia in pregnant women. i.v., intravenous; SVT, supraventricular tachycardia. [a]Adenosine 6–18 mg bolus. [b]Atenolol contraindicated. [c]Metoprolol 2.5–15 mg. [d]Verapamil 2.5–10 mg bolus over 5 min.

If these manoeuvres fail, i.v. adenosine (6–18 mg bolus) is recommended for termination of AV(N)RT. Intravenous beta-1-selective blockers (preferably metoprolol) can be administered for all SVTs, which either terminate the SVT or slow AV conduction and thereby the ventricular rate.[634] Intravenous beta-1-selective blockers (metoprolol), non-dihydropyridine CCBs (verapamil), and digoxin can be used safely.[635] In stable patients without structural heart disease, flecainide and ibutilide may be considered for termination of AF and AFL (*Figure 15*). In patients with congenital disease, synchronized, direct current cardioversion may be preferred.

12.4.1.2. Atrial fibrillation including anticoagulation

The incidence of arrhythmias among pregnant women is increasing, with AF being the most clinically relevant.[252,271,636] Atrial enlargement during pregnancy is accompanied by increased atrial function and both size and function of the left atrium reverse after normal pregnancy.[637] Atrial fibrillation is more frequent in pregnant women at older age, with high BMI[638], with ACHD, or with predisposing acquired conditions (e.g. hypertension, HF).[637] Compared to women <25 years of age, the odds ratio (OR) of AF episodes was 5.2 in women aged ≥40, and the OR was higher in the third compared to the first trimester.[638] Rapid atrioventricular conduction of AF may have serious haemodynamic consequences for both mother and foetus. Atrial fibrillation during pregnancy is associated with increased maternal death.[639] Atrial flutter in pregnant women most often occurs in the presence of ACHD or valvular heart disease (VHD) and in metabolic disturbances such as thyrotoxicosis or electrolyte disturbances.

Direct current cardioversion in pregnancy is required in haemodynamically unstable patients with monitoring of foetal heart rate (*Figure 15*).[250,268,271] Rhythm control is the preferred AF treatment strategy during pregnancy. For women with structurally normal hearts, anti-arrhythmic drugs (e.g. flecainide and sotalol, see *Section 5.2.5*) are not associated with foetal harm, although sotalol's beta-blocker effect necessitates monitoring of foetal growth.[250,268,271] There are minimal data on the use of propafenone during pregnancy, but this sodium channel blocker may be considered if flecainide is not available. Intravenous ibutilide or flecainide may be considered for termination of AFL and AF in haemodynamically stable patients.[640]

The indication for anticoagulation with LMWH before cardioversion or the need for transoesophageal echocardiography should be evaluated as in non-pregnant women and be maintained for at least 4 weeks after cardioversion.[636,641]

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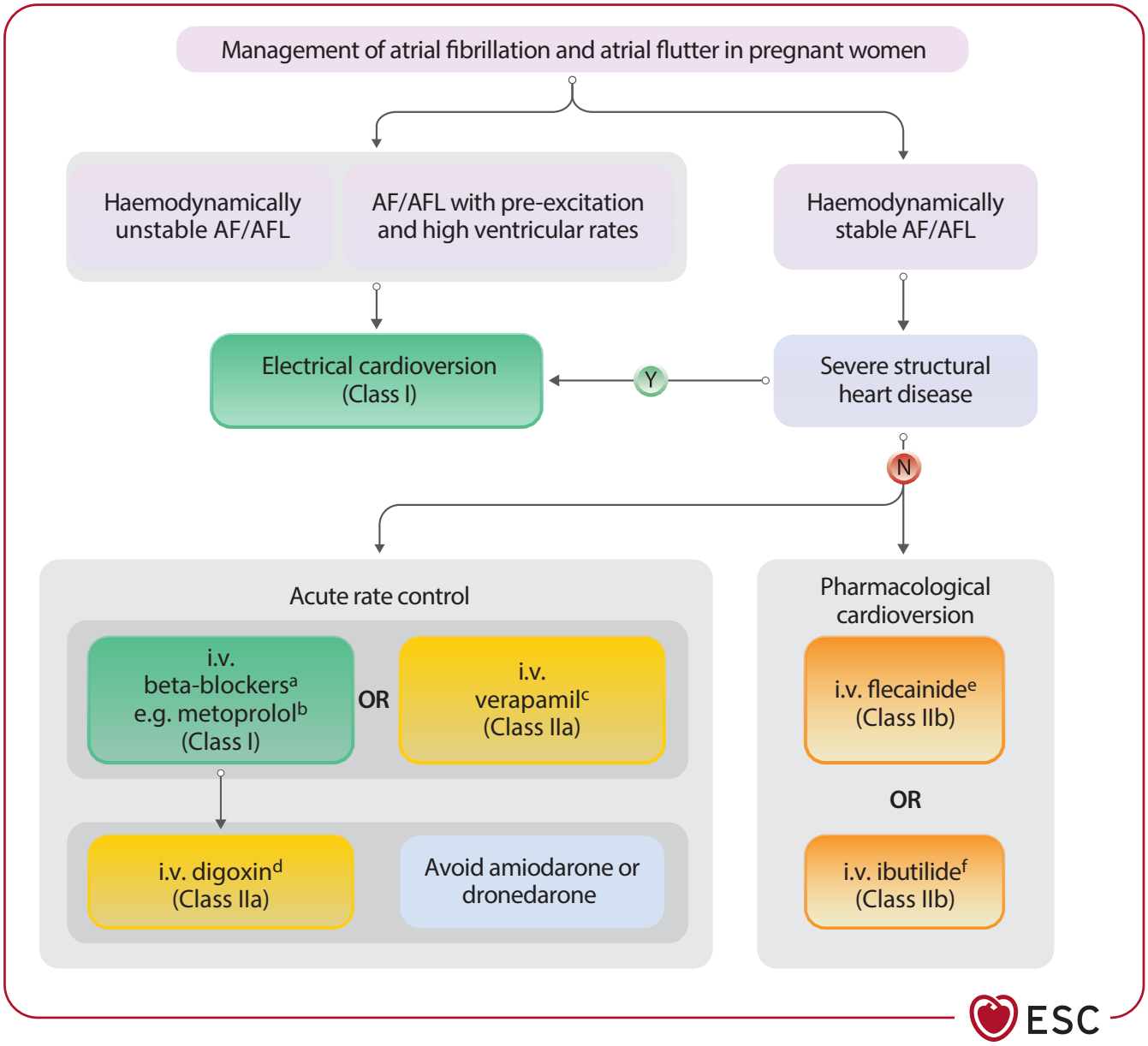

Figure 15 Management of atrial fibrillation and atrial flutter in pregnancy. AF, atrial fibrillation; AFL, atrial flutter; i.v., intravenous; N, no; SVT, supraventricular tachycardia; Y, yes. [a]Atenolol: contraindicated. [b]Metoprolol: 2.5–15 mg. [c]Verapamil: 2.5–1 mg bolus over 5 min. [d]Digoxin: 0.5 mg bolus, 0.75–1.5 mg over 24 h in divided doses. [e]Flecainide: 2 mg/kg over 10 min. [f]Ibutilide: <60 kg: 0.01 mg/kg over 10 min, repeated after 10 min if necessary; ≥60 kg: 1 mg over 10 min, repeated after 10 min if necessary.

When a rate control strategy is needed in the case of (long-standing) persistent or permanent AF, beta-blockers, verapamil, or digoxin should be used, also in combination, taking into consideration the concomitant conditions affecting the mother.[271,642]

Atrial fibrillation is a strong risk factor for cardioembolic events, and the hypercoagulable state of pregnancy may increase this risk. In pregnant women with persistent or permanent AF, the decision to anticoagulate is the same as in non-pregnant women and depends on the risk of thromboembolic events according to the CHA_2DS_2-VA score [congestive heart failure, hypertension, age ≥75 (doubled), diabetes, stroke (doubled), vascular disease, age 65–74].[643] The CHA_2DS_2-VA score threshold of ≥2 to anticoagulate has not been validated in pregnancy.[643,644] DOACs are contraindicated in pregnancy. The presence of mechanical valves or moderate to severe mitral stenosis require VKA (see *Section 5.2.1* and *Section 12.5*).

12.4.1.3. Pre-existing supraventricular tachycardia

In women with AVNRT, atrioventricular re-entry tachycardia (AVRT), and focal atrial tachycardia (FAT), chronic oral prophylaxis can be achieved with beta-blockers (metoprolol) or verapamil. For women with drug-refractory SVT or who have a contraindication to these drugs, flecainide or sotalol are reasonable alternatives, as is propafenone if flecainide is not available.

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In pregnant women with AVRT and Wolff–Parkinson–White (WPW) syndrome, arrhythmia episodes can be prevented by using oral flecainide, or propafenone when flecainide is not available. When AV-nodal blocking agents are used in WPW syndrome and AF occurs, the risk of rapid ventricular rates is increased. However, in pregnant women without documented AF, with known orthodromic AVRT, and with intermittent pre-excitation, long-term AV blockade is acceptable for prevention.

If catheter ablation treatment is necessary, it should be performed by experienced operators in a centre equipped with non-fluoroscopic mapping techniques (*Section 12.4.3.2*).

Recommendation Table 14 — Recommendations for supraventricular tachycardia and pregnancy

Acute management of SVT and AF	Class[a]	Level[b]
Immediate electrical cardioversion is recommended for acute treatment of SVT with haemodynamic instability.	I	C
Vagal manoeuvres and i.v. adenosine are recommended for conversion of haemodynamically stable supraventricular tachycardias.[645]	I	C
Intravenous beta-blockers[c] (e.g. metoprolol) are recommended as the first-line option for acute rate control in pregnant women with AF or AF with preserved LVEF and rapid ventricular rate.[646]	I	C
Intravenous digoxin or verapamil (if preserved LVEF) should be considered as a second-line option for initial rate control in pregnant women with AF or AFL and rapid ventricular rate.[635]	IIa	C
Ibutilide or flecainide may be considered for termination of AF and AFL in pregnant women without structural heart disease.[640,647]	IIb	C
Long-term management of SVT and AF		
Therapeutic anticoagulation with LMWH is recommended for pregnant women with persistent or permanent AF at elevated thromboembolic risk.[636,641]	I	C
Beta-1-selective blockers[c] are recommended for rate control in pregnant women with AF, AFL, or FAT.[146,271,648]	I	C
Beta-1-selective blockers[c] or verapamil are recommended for the prevention of SVT in women without pre-excitation on resting ECG.[146,268,649]	I	C
Flecainide or propafenone are recommended for the prevention of arrhythmias in pregnant women with WPW syndrome.[650]	I	C
Digoxin or verapamil should be considered for rate control in pregnant women with AF, AFL, or FAT when beta-blockers fail or are not tolerated.[146,271,645]	IIa	C
Flecainide, in addition to beta-blockers, should be considered for long-term AF rhythm control in pregnancy.[250,268,271,640]	IIa	C
Sotalol may be considered for rhythm management of AF and AFL with controlling for pro-arrhythmic risk factors as in non-pregnant women.[651]	IIb	C
Catheter ablation may be considered in pregnant women with recurrent, long symptomatic SVT, or with contraindications to pharmacological therapies.	IIb	C

AF, atrial fibrillation; AFL, atrial flutter; ECG, electrocardiogram; FAT, focal atrial tachycardia; i.v., intravenous; LMWH, low-molecular-weight heparin; LVEF, left ventricular ejection fraction; SVT, supraventricular tachycardia; WPW, Wolff–Parkinson–White.
[a]Class of recommendation.
[b]Level of evidence.
[c]Except for atenolol.

12.4.2. Ventricular arrhythmias

New-onset VT and ventricular fibrillation (VF) arising during pregnancy are rare (18 per 100 000 pregnancy-related hospitalizations).[652] The most common type of VT in pregnant women is idiopathic VT originating from the right ventricular outflow tract (RVOT) (*Figure 16*). When new-onset VT develops during the last 6 weeks of pregnancy or during the first month post-partum, underlying PPCM should be excluded.[370]

The use of amiodarone is not recommended in pregnancy and should be limited to women with refractory or life-threatening arrhythmias that cannot be controlled with any other anti-arrhythmic therapy. If administered, it requires close monitoring for potential side effects in the foetus, such as bradycardia or IUGR.

In women with known underlying substrates for VT, beta-blockers are recommended for prevention of VT.[148,252] In case of refractoriness or contraindications to beta-blockers, anti-arrhythmic therapy with flecainide, sotalol, or quinidine is recommended, with the choice of drug based on the underlying cardiac substrate (*Figure 16*).[148,252] Idiopathic RVOT-VT can be prevented with beta-blockers or verapamil.[653] When this is ineffective, flecainide[654] or sotalol[650,655–659] are safe options to consider for prophylaxis of idiopathic RVOT-VT.

Recommendations on the acute management of ventricular arrhythmias during pregnancy are described in the 2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death, and are depicted in *Figure 16*.[252]

12.4.3. Cardioversion, ablation, and device implantation, and implantable cardioverter defibrillator management

12.4.3.1. Electrical cardioversion

Electrical cardioversion is safe and effective in all pregnancy phases as it does not affect foetal circulation or induce foetal arrhythmia. In pregnancy there are no changes in transthoracic impedance compared to non-pregnant women,[660] and shock energies delivered should therefore be the same as in non-pregnant patients. The foetal heart rate should be monitored after cardioversion.[631,632,661,662]

12.4.3.2. Catheter ablation

Catheter ablation in pregnant women should preferably be performed after the first trimester in a centre with experience in non-fluoroscopic

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electro-anatomical mapping and catheter navigation systems.[663–665] Catheter ablation for drug-refractory and poorly tolerated VT or for recurrent drug-refractory AVNRT, AVRT, FAT, cavotricuspid isthmus-dependent AFL, and certain benign right-sided VTs may be considered to avoid potentially harmful anti-arrhythmic drug effects during pregnancy, although it has no role in AF.[664,666]

12.4.3.3. Device implantation

An ICD is indicated in pregnant women who are at high risk of sudden cardiac death.[252] Implantation of a device is not associated with a higher risk of major complications and can be performed safely,[667,668] particularly after 8 weeks of gestation. During implantation, radiation exposure should be minimized following the ALARA principle (see

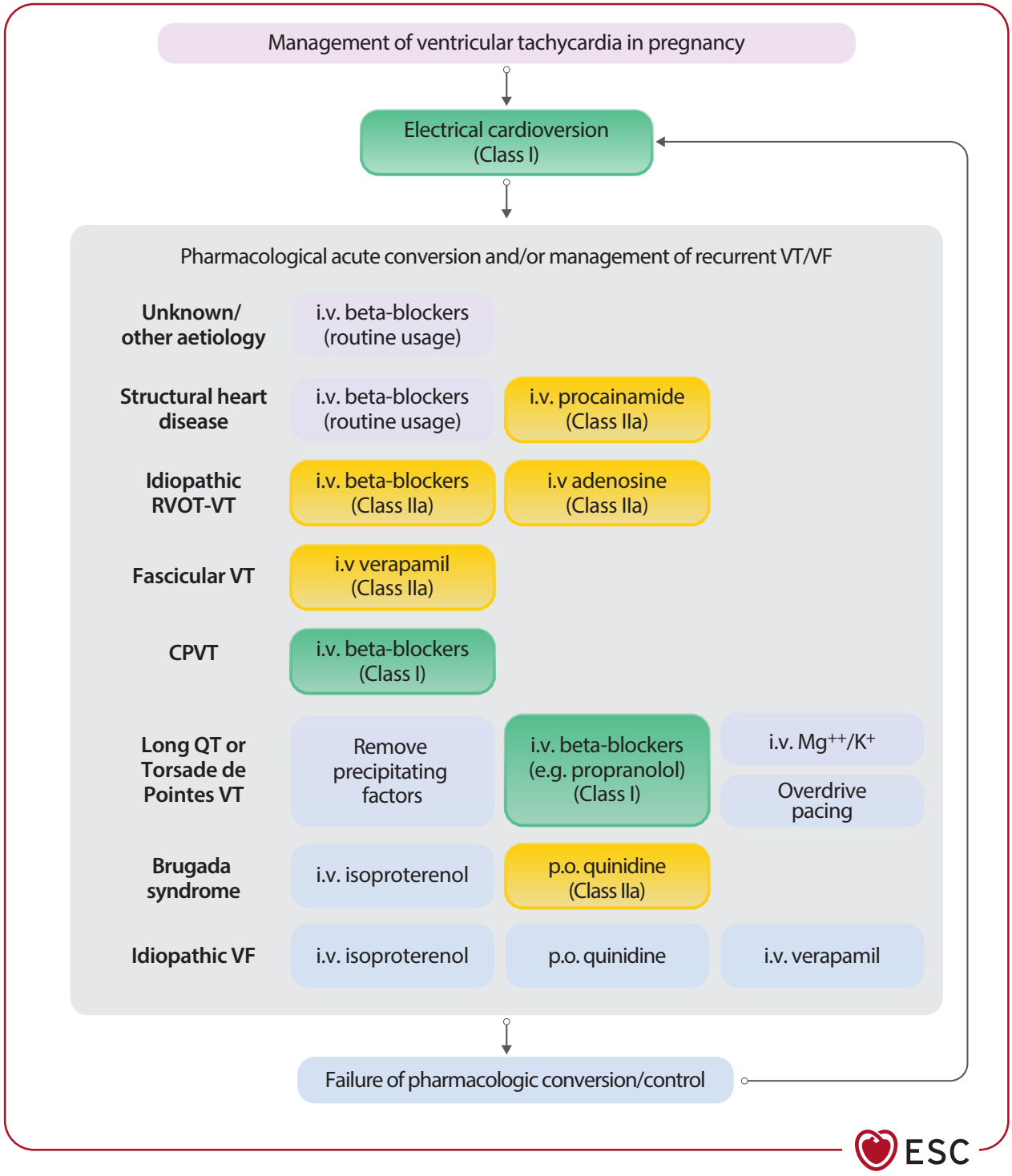

Figure 16 Management of ventricular tachycardias in pregnancy. CPVT, catecholaminergic polymorphic ventricular tachycardia; i.v., intravenous; N, no; RVOT, right ventricular outflow tract; VF, ventricular fibrillation; VT, ventricular tachycardia; Y, yes. See Supplementary data online (*Table S6*) for medication dosages. Adapted from Zeppenfeld *et al.*[252].

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Section 4.3.5) or with non-fluoroscopic imaging techniques.[669] Women with an ICD should maintain their regular ICD care throughout pregnancy.[669]

12.4.3.4. Implantable cardioverter defibrillator management

Routine ICD interrogation and guidance are recommended prior to delivery. Management of pregnant women with an ICD or pacemaker is summarized in *Figures 17* and *18*.

Women with structural heart disease are at higher risk of atrial and ventricular tachyarrhythmias during pregnancy.[670] Consequently, pregnant women with ICDs may present with either an inappropriate shock (due to e.g. lead defects, new-onset supraventricular tachyarrhythmias, or technical issues such as T-wave oversensing or noise sensing) or an appropriate ICD shock caused by ventricular tachyarrhythmias. In case of tachyarrhythmia, triggering events such as HF, electrolyte abnormalities, ischaemia, or infectious disease should be ruled out, as with non-pregnant women. Depending on the underlying cause, therapy consists of device reprogramming or initiating or adapting anti-arrhythmic drug therapy. In the case of drug-refractory arrhythmias or serious anti-arrhythmic drug side effects, catheter ablation may be considered in experienced centres.[664]

Most data indicate that maternal ICD shocks do not have major foetal adverse effects.[668,671] The approach to pregnant women presenting with ICD shocks is no different from non-pregnant women (*Figure 18*).[252]

A WCD may be considered when pregnant women have an ICD indication from reversible conditions.[365,672–674]

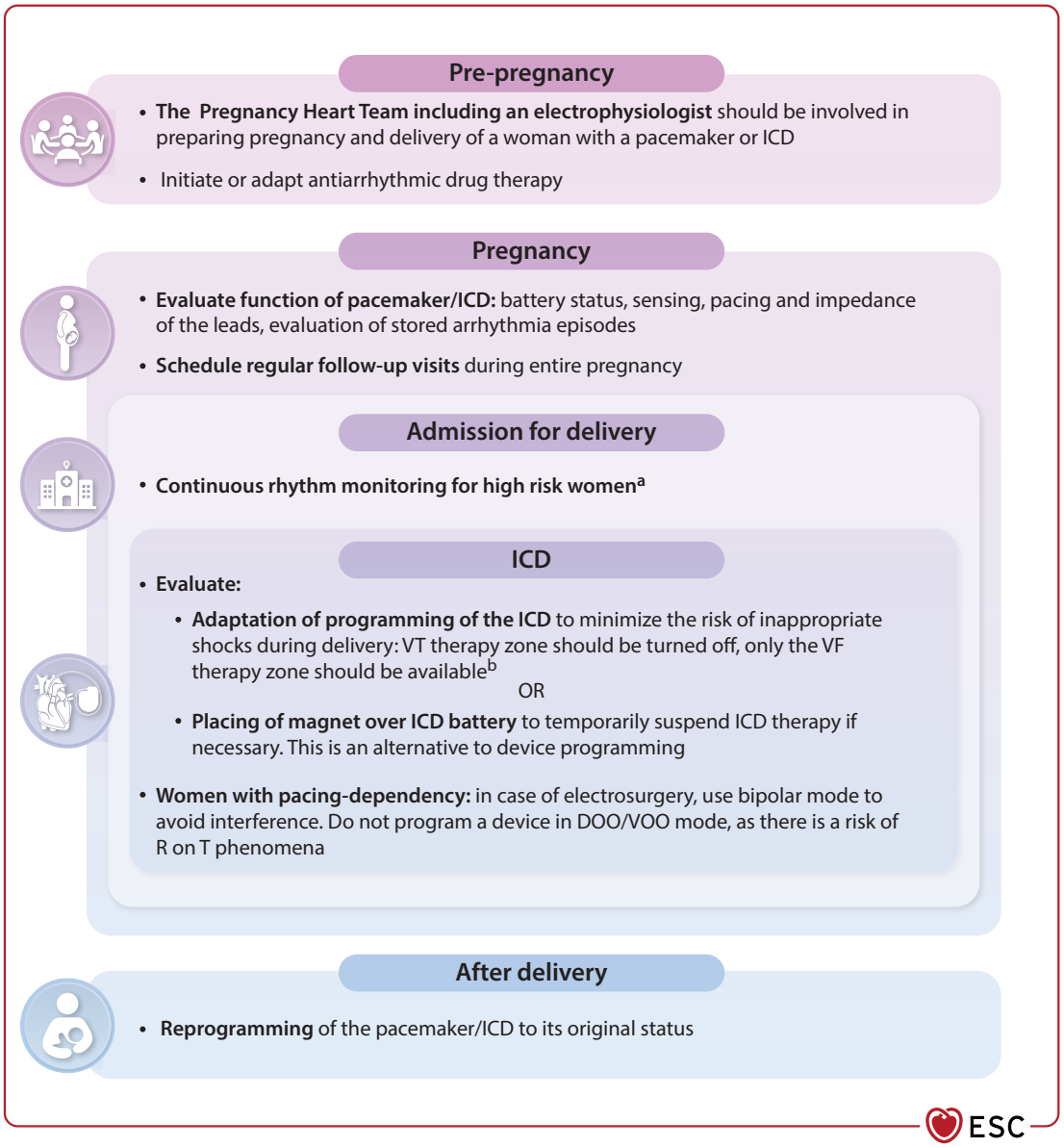
Pre-pregnancy
• The Pregnancy Heart Team including an electrophysiologist should be involved in preparing pregnancy and delivery of a woman with a pacemaker or ICD
• Initiate or adapt antiarrhythmic drug therapy
Pregnancy
• Evaluate function of pacemaker/ICD: battery status, sensing, pacing and impedance of the leads, evaluation of stored arrhythmia episodes
• Schedule regular follow-up visits during entire pregnancy
Admission for delivery
• Continuous rhythm monitoring for high risk women[a]
ICD
• Evaluate:
• Adaptation of programming of the ICD to minimize the risk of inappropriate shocks during delivery: VT therapy zone should be turned off, only the VF therapy zone should be available[b]
OR
• Placing of magnet over ICD battery to temporarily suspend ICD therapy if necessary. This is an alternative to device programming
• Women with pacing-dependency: in case of electrosurgery, use bipolar mode to avoid interference. Do not program a device in DOO/VOO mode, as there is a risk of R on T phenomena
After delivery
• Reprogramming of the pacemaker/ICD to its original status
ESC

Figure 17 Management of pacemaker and implantable cardioverter defibrillator and pregnancy. b.p.m., beats per minute; ICD, implantable cardioverter defibrillator; VF, ventricular fibrillation; VT, ventricular tachycardia. [a]Patients with an increase in arrhythmias during pregnancy, history of ventricular tachyarrhythmias. [b]Single zone configuration: VT detection at 250 b.p.m., prolonged detection duration, e.g. 30 out of 40 beats.

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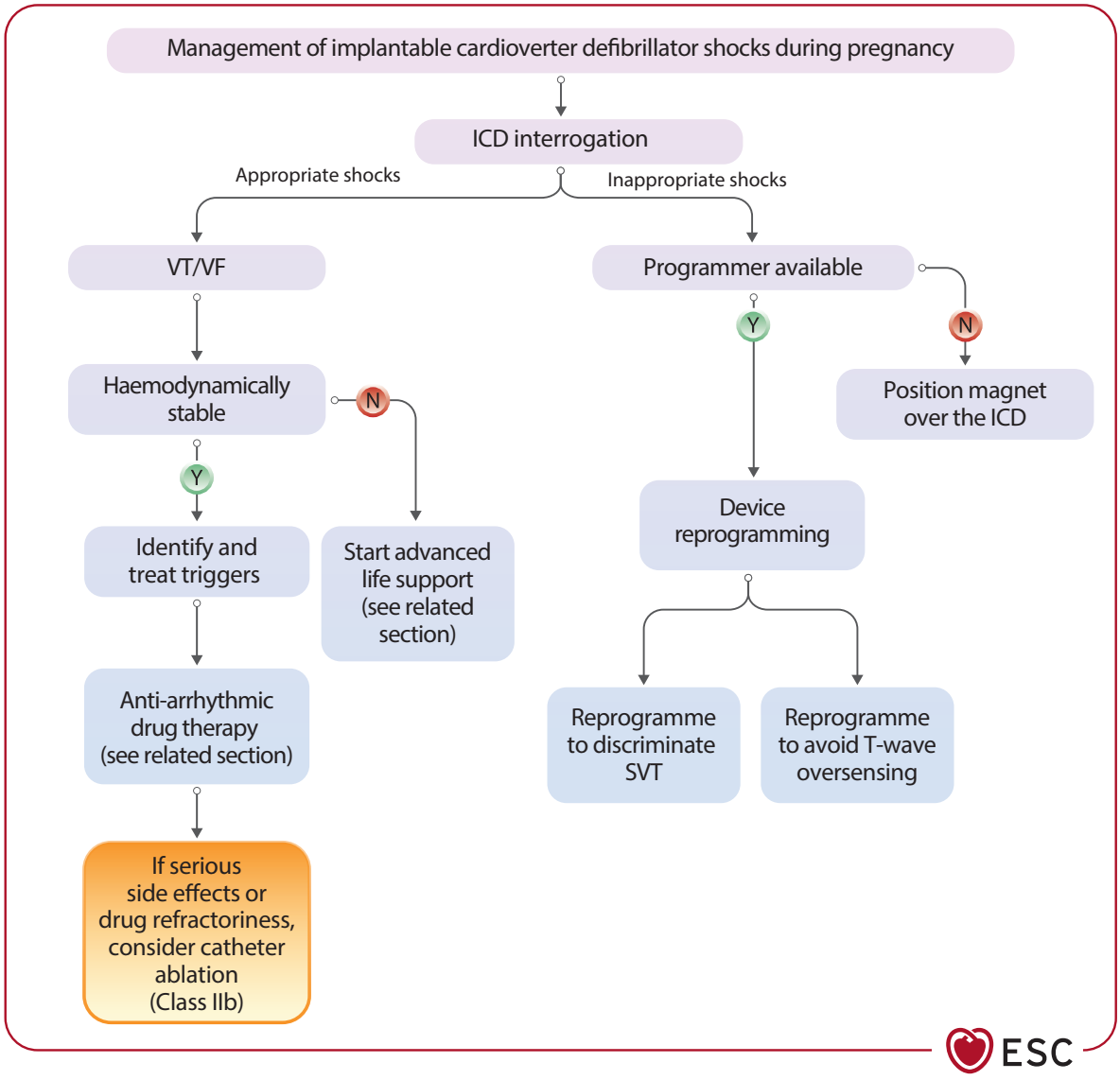

Figure 18 Management of implantable cardioverter defibrillator shocks in pregnancy. ICD, implantable cardioverter defibrillator; N, no; SVT, supraventricular tachycardia; VF, ventricular fibrillation; VT, ventricular tachycardia; Y, yes.

Recommendation Table 15 — Recommendations for ventricular tachycardia, device implantation, catheter ablation, and pregnancy

Recommendations	Class[a]	Level[b]
Immediate electrical cardioversion is recommended for both unstable and stable ventricular tachycardias.[252]	I	C
Beta-blockers or verapamil are recommended for the prevention of idiopathic sustained VT.	I	C
If an ICD, pacemaker, or resynchronization therapy device is indicated during pregnancy, implantation is recommended with optimal radiation protection.[667,675]	I	C
In idiopathic RVOT-VT, flecainide should be considered if beta-blockers fail, to prevent recurrence.	IIa	C
For acute conversion of haemodynamically stable sustained VTs during pregnancy, i.v. beta-blocker, adenosine (idiopathic RVOT-VT), verapamil (fascicular VT), procainamide, or overdrive ventricular pacing (ICD lead) should be considered.[252,653,676–678]	IIa	C
When performing catheter ablation during pregnancy, the use of non-fluoroscopic mapping and navigation systems should be considered.[663–665]	IIa	C
Catheter ablation with electro-anatomical mapping systems may be considered in experienced centres in the case of sustained drug-refractory, recurrent, and/or poorly tolerated VT if there are no other alternatives.	IIb	C

Continued

ICD, implantable cardioverter defibrillator; i.v., intravenous; RVOT, right ventricular outflow tract; VT ventricular tachycardia.
[a]Class of recommendation.
[b]Level of evidence.

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12.4.4. Cardiac arrest

Maternal cardiac arrest occurs in 8/100 000 hospitalizations in the United States of America[679] and 7.6/100 000 pregnancies in the Netherlands.[680] Haemorrhage and anaesthetic complications are the most common overall causes of cardiac arrest,[681] and HF, ACS, arrhythmias, aortic dissection, and PE are the most common cardiovascular causes of cardiac arrest.[682,683]

Because most of the underlying causes of cardiac arrest in pregnancy tend to be reversible, pregnant women have better outcomes than non-pregnant women.[684] Basic cardiac arrest resuscitation principles apply to pregnant women, although some differences should be considered.[684] If cardiac arrest occurs beyond 20 weeks of pregnancy, left lateral manual displacement of the uterus or left lateral position of the woman is indicated to avoid aortocaval compression (*Figure 19*).[684,685] Chest compressions should be according to basic life support guidelines.

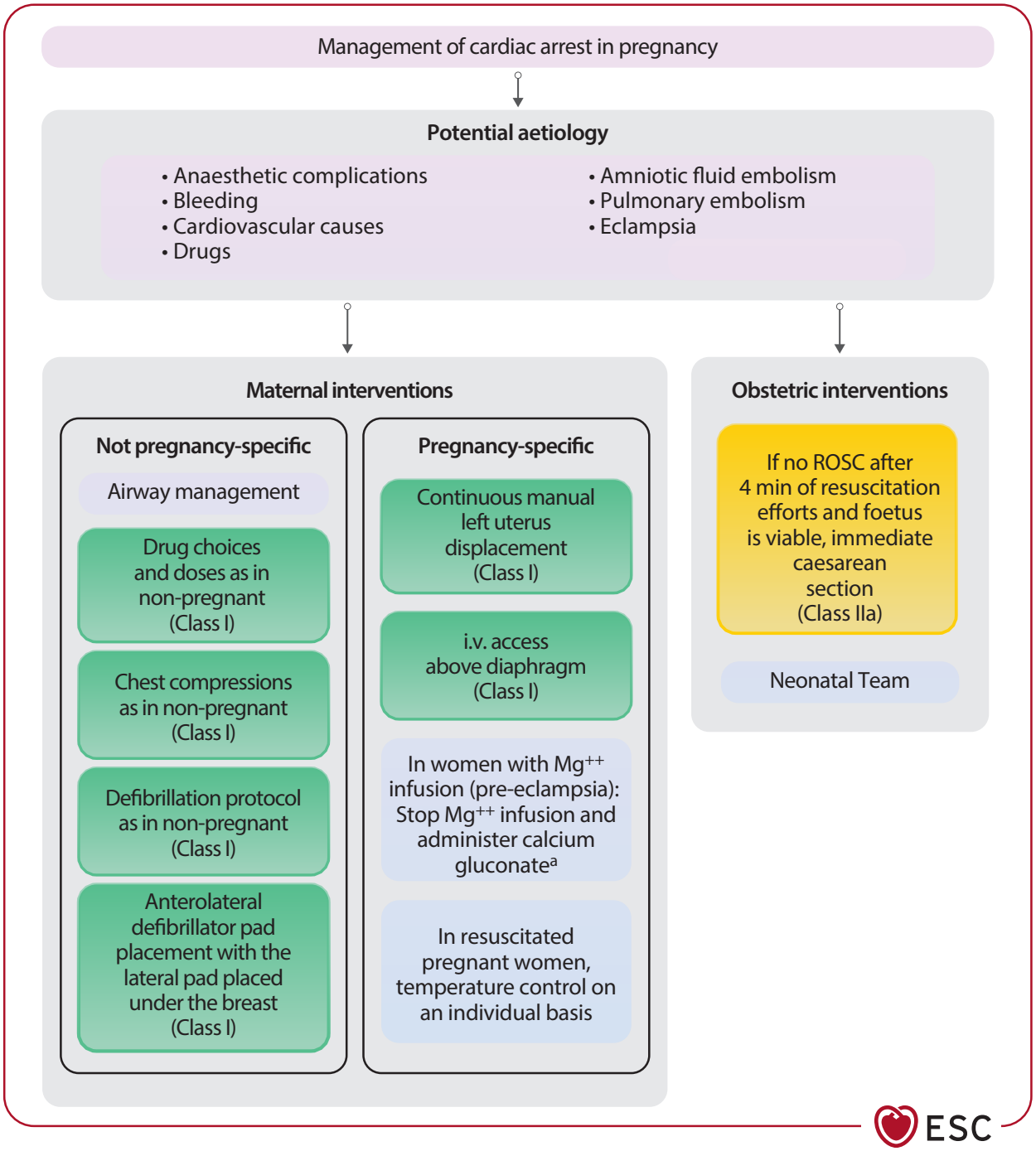
Management of cardiac arrest in pregnancy
Potential aetiology
• Anaesthetic complications
• Bleeding
• Cardiovascular causes
• Drugs
• Amniotic fluid embolism
• Pulmonary embolism
• Eclampsia
Maternal interventions
Not pregnancy-specific
Airway management
Drug choices and doses as in non-pregnant (Class I)
Chest compressions as in non-pregnant (Class I)
Defibrillation protocol as in non-pregnant (Class I)
Anterolateral defibrillator pad placement with the lateral pad placed under the breast (Class I)
Pregnancy-specific
Continuous manual left uterus displacement (Class I)
i.v. access above diaphragm (Class I)
In women with Mg++ infusion (pre-eclampsia): Stop Mg++ infusion and administer calcium gluconate[a]
In resuscitated pregnant women, temperature control on an individual basis
Obstetric interventions
If no ROSC after 4 min of resuscitation efforts and foetus is viable, immediate caesarean section (Class IIa)
Neonatal Team
ESC

Figure 19 Management of cardiac arrest in pregnancy. i.v., intravenous; ROSC, return of spontaneous circulation. [a]30 mL calcium gluconate 10% solution.

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In post-arrest care, the full left lateral decubitus position is recommended. The use of device-assisted chest compressions in pregnancy is currently not recommended due to lack of data.[684] Importantly, no drugs should be withheld because of concerns about teratogenicity.[614,686] Foetal monitoring after cardiac arrest is mandatory.

Emergency caesarean section should be immediately prepared for and considered when initial resuscitation fails.[682] If this is not feasible, rapid maternal transfer is advised to an appropriate clinical setting with uninterrupted resuscitation.

Recommendation Table 16 — Recommendations for cardiac arrest and pregnancy

Recommendation	Class[a]	Level[b]
Continuous manual left uterine displacement during CPR in pregnant women (≥20 weeks) with cardiac arrest is recommended to relieve aortocaval compression.[614]	I	C
It is recommended to establish i.v. access above the diaphragm to ensure that the i.v. therapy is not obstructed by the gravid uterus.[614]	I	C
It is recommended to perform the same chest compressions and defibrillation protocols in pregnant as in non-pregnant women.[554,614,687]	I	C
Anterolateral defibrillator pad placement is recommended with the lateral pad placed under the breast.[614,660]	I	C
It is recommended that no drugs are withheld in pregnant women with cardiac arrest due to concerns of teratogenicity.[686]	I	C
Immediate caesarean section at the site of the arrest should be considered and immediately prepared if ROSC has not been achieved in the mother after 4 min of resuscitative efforts and if the foetus is viable, taking gestational age, comorbidities, and the available level of medical care into account.[614,688,689]	IIa	C

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CPR, cardiopulmonary resuscitation; i.v, intravenous; ROSC, return of spontaneous circulation.
[a]Class of recommendation.
[b]Level of evidence.

12.4.5. Bradycardia

12.4.5.1. Sinus node dysfunction

Sinus bradycardia due to sinus node dysfunction is uncommon in pregnant women who do not have structural heart disease. In the second trimester, symptomatic bradycardia may occur as a result of a reduction in systemic resistance, but this rarely requires treatment. Symptomatic bradycardia during pregnancy may be caused by the supine hypotensive syndrome, defined as a systolic BP decrease of >15 mmHg due to compression of the inferior vena cava by the uterus.[690] Management depends on the underlying cause, severity of symptoms, and potential risks to both the mother and the foetus.

12.4.5.2. Atrioventricular block

Mobitz type I AV block is common in pregnant women and rarely progresses during pregnancy.[691] There are no data on progression of congenital AV conduction block during pregnancy and vaginal delivery does not cause extra risk for mothers who are asymptomatic, haemodynamically stable, and have a normal cardiac anatomy and function.[691] Prophylactic placement of temporary pacemaker wires is not usually indicated but is an individualized decision.

12.4.5.3. Management of sinus node dysfunction and atrioventricular block

In acute, life-threatening settings, bradycardia should be treated as in non-pregnancy. Isoproterenol can be used when benefits outweigh risks. It is not known if it is excreted in human milk. Pacing indications (temporary and permanent) do not differ between pregnant and non-pregnant women and can be found in the 2021 ESC Guidelines on cardiac pacing and cardiac resynchronization therapy.[692] Pacemakers can be implanted safely during pregnancy using standard methods with minimal fluoroscopy or non-fluoroscopic methods.[675,693–695]

Recommendation Table 17 — Recommendation for congenital atrioventricular block and pregnancy

Recommendation	Class[a]	Level[b]
In pregnant women with asymptomatic congenital AV block, normal cardiac anatomy and function, a narrow QRS complex, and ventricular rate (≥50 b.p.m.), a prophylactic temporary pacemaker during delivery is not recommended.[691]	III	C

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AV, atrioventricular; b.p.m., beats per minute; QRS, Q, R, and S waves.
[a]Class of recommendation.
[b]Level of evidence.

12.5. Valvular heart disease

During childbearing years VHD is usually either congenital or rheumatic in cause. Complications during pregnancy include new-onset AF, which can lead to deterioration in cardiac function, an increased risk of thromboembolic events, and heart failure.[696,697] Women with valve disease should have access to pre-conception counselling and their cardiac condition should be optimized. This may include pre-pregnancy intervention or surgery for those with symptoms or severe disease (*Figure 20*), in keeping with the 2021 ESC/EACTS Guidelines for the management of valvular heart disease.[698] Dedicated tools for risk stratification may be needed in the setting of rheumatic heart disease.[697]

12.5.1. Stenotic native valve lesions

Stenotic valve lesions limit the ability to increase CO during pregnancy. The result of this may be presentation with symptoms for the first time during pregnancy. Serial TTE in pregnancy will usually demonstrate an increase in valve gradient of up to 50% due to the normal pregnancy-related increase in CO.[699]

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12.5.1.1. Mitral valve stenosis

Mitral valve stenosis is a common cause of HF during pregnancy.[11] Mild stenosis is usually well tolerated.[11] However, symptoms may occur if the valve area is <1.5 cm^2. Maternal mortality in mitral stenosis is higher in those with NYHA >II, systolic pulmonary arterial pressure (PAP) >30 mmHg, severe stenosis, older age, and in low-income countries[11,696,700] (*Figure 20*). Foetal risks include increased rates of premature delivery, IUGR, and foetal death, especially in highly symptomatic mothers (NYHA III/IV).[701]

12.5.1.1.1. Management. Diagnosis is as per usual criteria.[698] During pregnancy, valve area by 2D-planimetry is thought to be more reliable than flow-dependent measures, because higher stroke volume and tachycardia will increase the measured gradient across the valve.[702] Mitral valve anatomy, especially the presence of subvalvar involvement or the presence of MR, is important if considering intervention.

If signs or symptoms of PH are present, activity restriction should be suggested, and beta-blockers and/or diuretics should be started. Anticoagulation regimens should be individualized. Therapeutic anticoagulation with full therapeutic-dose LMWH or VKA (see *Section 5*) is indicated in those with AF, left atrial clot, or a previous embolism. Anticoagulation should be considered in those with significant mitral stenosis, spontaneous echo contrast in the left atrium, dilated left atrium with left atrial volume index >60 mL/mL^2, or those in HF.

12.5.1.1.2. Interventions. Pre-pregnancy intervention should be considered in those with significant stenosis (valve area <1.5 cm^2). Before and during pregnancy, percutaneous mitral balloon commissurotomy is the primary intervention in those who remain in NYHA III/IV or with severe PAP elevation despite medical therapy.[703] Balloon techniques are highly successful during pregnancy unless there is complex subvalvar involvement. Closed commissurotomy is rarely used but is an alternative. Open valve surgery should only be used when there is a risk to maternal life and other options are not possible or have failed.[704]

12.5.1.1.3. Delivery. Vaginal delivery is the preferred option. Caesarean section is preferred in cases of severe mitral stenosis and for those with refractory HF. Delivery is a time of increased risk of HF and thrombotic events.[182]

12.5.1.1.4. Post-partum. Close monitoring is needed in the days following delivery and diuretics may be required to treat fluid overload. The long-term outcome depends on the risk of valve progression and the success of commissurotomy if performed. Lifelong follow-up is required.

12.5.1.2. Valvular aortic stenosis

Bicuspid aortic valve disease is a common cause of valvular aortic stenosis in women of childbearing age. The outcome is related to the baseline severity of the stenotic lesion. Women with severe stenosis and those with symptoms prior to pregnancy have a 1 in 4 risk of developing HF during pregnancy.[703] Exercise testing and measurement of NP can be used to stratify risk prior to pregnancy. Those with symptomatic severe aortic stenosis should be offered intervention prior to pregnancy. Risk-stratifying those with asymptomatic aortic stenosis is more challenging. Left ventricular systolic dysfunction, or effort limitation on exercise testing, indicate that intervention before pregnancy should be considered.[698]

Women with severe aortic stenosis contemplating pregnancy should be counselled regarding the risks and offered surgery if they prefer.

Despite this, maternal mortality is rare for those under expert care.[703] The risk of sudden death in those with severe stenosis is increased but difficult to quantify. BAV-associated aortopathy is discussed in *Section 8.3*.

12.5.1.2.1. Management. Medical therapy has a limited role in symptomatic aortic stenosis. Diuretics may help those with HF or high filling pressures, but caution should be exercised. In women with severe symptomatic aortic stenosis, rest and possibly hospital admission should be considered. Intervention should be considered in women with persisting symptoms including angina or with new ST changes on ECG. In symptomatic pregnant women with severe aortic stenosis not responding to medical therapy, non-surgical options such as balloon valvuloplasty or transcatheter aortic valve implantation (TAVI) may be considered during pregnancy.[705,706] Procedures should be performed in an experienced valve centre. If no catheter-based options are available, surgical valve replacement or repair is recommended. If the foetus is at a viable gestation, taking account of other comorbidities and the available level of neonatal care, delivery should occur prior to valve intervention. These complex decisions should be discussed with the full Pregnancy Heart Team.[707] Cardiac surgery with cardiopulmonary bypass is associated with at least a 20% risk of foetal loss.[708]

Vaginal delivery is the preferred mode of delivery for the majority of women. In those with severe symptomatic aortic stenosis, caesarean delivery should be considered. Early post-partum HF may develop.

For pulmonary stenosis, see *Section 9* on congenital heart disease.

12.5.2. Regurgitant native valve lesions

Valve regurgitation is generally better tolerated than valve stenosis in pregnancy. Increased maternal and foetal event rates can be seen in those with severe regurgitation.[709]

12.5.2.1. Mitral and aortic valve regurgitation

Women with valve regurgitation and either symptoms or LV dysfunction incur an increased risk of HF, occurring in 20%–25% of those with at least moderate regurgitation.[182,710] Acute regurgitant lesions are often less well tolerated than chronic regurgitation.[710]

12.5.2.1.1. Management. Diuretics can be used in those with severe symptomatic mitral or aortic regurgitation. Cardiac surgery is rarely required during pregnancy. Vaginal delivery is preferred unless the mother is in refractory HF.

12.5.2.2. Arrhythmogenic mitral valve prolapse

Arrhythmic mitral valve prolapse (AMVP) is defined as the presence of mitral valve prolapse and arrhythmia symptoms or signs, such as frequent ventricular premature contractions or complex ventricular arrhythmias.[711] The arrhythmias may arise from myocardial fibrosis resulting from prolonged and increased stretch of papillary muscles and of the inferolateral LV wall by the prolapsing valves.

Several reports have included female sex as a risk factor for severe arrhythmic events.[711] Other risk factors include syncope, the presence of mitral annular disjunction, late gadolinium enhancement on CMR, complex ventricular arrhythmias, including non-sustained ventricular tachycardia, syncope, T-wave inversions in the inferolateral ECG leads, bi-leaflet mitral valve prolapse, and reduced LVEF. Little is known about the effect of pregnancy in women with AMVP, but altered loading conditions may lead to increased tension in mitral

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valve apparatus and thereby increased risk of ventricular arrhythmias. A recent publication indicated increased arrhythmic risk during pregnancy in women with AMVP and life-threatening ventricular arrhythmias.[712]

It is recommended that women with AMVP undergo pre-conception counselling and optimization of medication. Medical treatment includes anti-arrhythmic therapy with, for example, beta-blockers and/or flecainide.[713] If started pre-pregnancy, treatment should continue during pregnancy with close follow-up and arrhythmic monitoring. A pregnant woman with AMVP should be monitored during pregnancy at a tertiary centre with experience in AMVP and with periodic Holter monitoring to identify a potential increase in arrhythmic burden.[711] Risk stratification for ICD implantation should follow the consensus for AMVP in general.[711]

12.5.2.3. Tricuspid regurgitation

Medical treatment for tricuspid regurgitation is usually not required but during pregnancy diuretics and medication for rhythm management may be needed. Surgery for isolated TR in pregnancy is rarely indicated except for the setting of endocarditis.

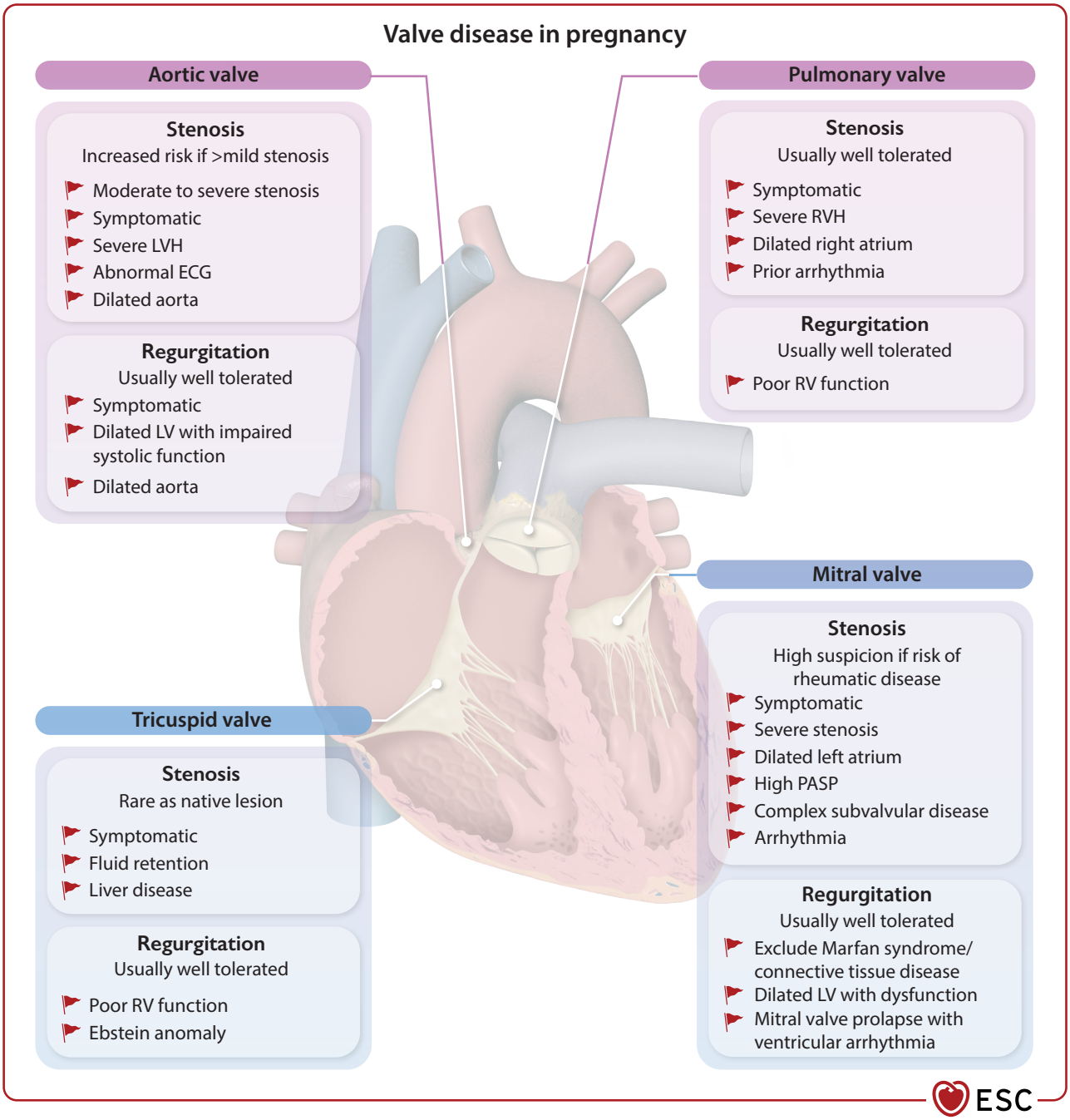

Figure 20 Valvular heart disease and pregnancy. ECG, electrocardiogram; LV, left ventricle; LVH, left ventricular hypertrophy; PASP, pulmonary arterial systolic pressure; RV, right ventricle; RVH, right ventricular hypertrophy. Red flags indicate high-risk features.

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Recommendation Table 18 — Recommendations for native valve disease and pregnancy

Recommendations	Class[a]	Level[b]
Intervention is recommended before pregnancy in symptomatic patients with severe aortic stenosis.[698]	I	C
Intervention is recommended before pregnancy in women with mitral stenosis and a valve area <1.5 cm^2. [698,701,714]	I	C
In pregnant women with symptomatic mitral stenosis or pulmonary hypertension, restricted activities and beta-blockers are recommended.[20]	I	C
In pregnant women with mitral stenosis, diuretics are recommended when congestive symptoms persist despite beta-blockers.[20]	I	C
Full therapeutic-dose anticoagulation is recommended in women with mitral stenosis complicated by AF, left atrial thrombus, or prior embolism.	I	C
Surgical treatment is recommended before pregnancy in women with severe aortic or mitral regurgitation with symptoms, impaired ventricular function, or marked ventricular dilatation.[698,715]	I	C
Diuretics are recommended in pregnant women with regurgitant lesions when symptoms or signs of congestion occur.	I	C
Intervention should be considered before pregnancy in those with asymptomatic severe aortic stenosis after counselling on the risks and benefits.[698]	IIa	C
Percutaneous mitral commissurotomy for mitral stenosis should be considered in pregnant women with severe symptoms or systolic pulmonary artery pressure >50 mmHg despite medical therapy.[698]	IIa	C
Valve surgery during pregnancy should only be considered when there is a maternal mortality risk and other treatment options have failed.[421]	IIa	C
In very selected symptomatic pregnant women with severe aortic stenosis not responding to medical therapy, non-surgical options such as balloon valvuloplasty or TAVI may be considered.[716]	IIb	C

AF, atrial fibrillation; TAVI, transcatheter aortic valve implantation.
[a]Class of recommendation.
[b]Level of evidence.

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12.5.3. Prosthetic valves

When a woman of childbearing age or a girl requires valve surgery, careful consideration should be given to the possibility of future pregnancies. When appropriate, the discussion regarding valve choice should involve the Pregnancy Heart Team. In general, valve repair, valve-in-valve, or non-mechanical valves are preferable, avoiding the need for anticoagulation. Data from the ROPAC III study, focusing on prosthetic valves, show that the chance of an uncomplicated pregnancy with a live birth in women with a MHV was 54%, compared to 79% in women with a tissue valve.[177] Regional differences, especially between high-income and low- and middle-income countries, need to be acknowledged, as also indicated in a recent study from Madras in India (M-PAC registry) describing pregnancy outcomes in 70 women with a prosthetic heart valve.[717] A very high foetal death rate and major adverse cardiovascular events (MACE) rate (40% and 34%, respectively),[717] including a high number of valve thromboses, is believed to relate not only to differences in organization of care, but also to underlying valvular disease, with more rheumatic heart disease in low- and middle-income countries. The Ross procedure in aortic valve disease is an alternative option to be considered. In the absence of aortic dilatation, pregnancy risk is low after the Ross procedure.[718]

12.5.3.1. Bioprosthetic valves

Prior to pregnancy, a full assessment of valve function should be performed. Many women are on lifetime low-dose aspirin treatment, and this should be continued during pregnancy unless there is a contraindication. If there is severe valve dysfunction prior to pregnancy, reintervention should be considered. However, in this setting the risk of pregnancy with the current valve dysfunction should be balanced against the risk of the new valve being proposed. Mechanical valve implantation should be avoided when possible. Transcatheter valve-in-valve intervention may have a role in extending the lifespan of a failing bioprosthetic valve in a young woman contemplating pregnancy. In a recent retrospective study of CARPREG data, 215 pregnancies in 101 women who had implanted bioprosthetic valves prior to pregnancy were described. More than a quarter had some degree of valvar dysfunction, although the time since the last valve replacement surgery was only 6 ± 3 years on average. Bioprosthetic valve dysfunction was more than twice as common in women with left-sided as opposed to right-sided valves.[719]

The chance of a pregnancy without serious adverse events and a live birth in women with a bioprosthetic valve is 79%.[177] When significant bioprosthetic dysfunction is present, the risk of complications can be significant, especially if associated with severe stenosis or ventricular dysfunction. There is no compelling evidence that pregnancy is associated with accelerated valve deterioration.

12.5.3.2. Mechanical valves

Pregnant women with MHVs are exposed to a high risk of complications (mWHO 2.0 risk classification ≥III). The chances of an event-free pregnancy with a live birth were only 58% in the initial ROPAC II study and showed no improvement after 8 years in the ROPAC III study (54%).[177,204] Thrombotic complications occur in 9%–24% and bleeding complications in 20%–30% of the cases in the ROPAC III and a United Kingdom study, respectively.[176,177] Women with a mechanical valve in the mitral position are especially at risk of adverse outcomes, including mortality.[177]

All women with MHVs should be fully counselled pre-pregnancy regarding the risks and benefits of the various anticoagulation regimens. When planning the optimal anticoagulation strategy, logistical issues,

such as access to timely anti-factor Xa level testing and maternal ability to adhere to treatment regimens, need to be considered.

12.5.3.2.1. Anticoagulation during pregnancy. The most effective regimen in preventing maternal thrombotic complications is the continuous use of VKAs. Thrombotic events not only compromise the mother but also jeopardize the baby.[170,200,204,206,720,721] However, there remain concerns about the foetal impact of VKAs. Data from the ROPAC III study indicated a higher risk of miscarriage in VKA users.[177] As discussed in *Section 5*, the foetal risk of VKAs is in part dose-related. Although there is no safe dose for the foetus, event rates are reduced when lower doses of VKA are used.[200] For these reasons, continuation of VKAs should be considered when the risk of thrombosis is high and the dose required to achieve the target INR is low (see *Section 5*). Target INRs are unchanged from non-pregnant values.[698] INR monitoring should be weekly or every 2 weeks (see *Section 5*).

The alternative strategy is switching to therapeutic-dose LMWH (twice daily) until the 12th week of pregnancy with a monitoring plan. The target peak levels of anti-factor Xa should be discussed individually and vary between 1 and 1.2 IU/mL. The value of trough anti-factor Xa levels is less clear. Adjusting dosing to obtain levels of >0.6 IU/mL has been suggested, however, with little evidence.[170,216] Dosing regimens for therapeutic LMWH are provided in *Section 5*. In women with a very high thrombotic risk, the addition of low-dose acetylsalicylic acid (ASA) should be considered.[170] In rare circumstances, such as the unavailability of anti-factor Xa level testing, i.v. UFH can be used. Target aPTT levels with a prolongation of ≥2 times the control can be challenging to achieve and may require multiple adjustments of dosing and prolonged inpatient care. In the majority of cases, VKAs will be the favoured therapy in the second and third trimesters to minimize the maternal risks after the period of embryogenesis.[176,204] Despite this, some women will choose to remain on therapeutic-dose LMWH throughout pregnancy. The management of anticoagulants during pregnancy in women with MHVs is summarized in *Figure 21*.

12.5.3.2.2. Mechanical valve thrombosis. Regular maternal cardiac ultrasound should be performed during pregnancy to assess valve function and rule out valve thrombosis. Imaging once per trimester will usually suffice unless there is pre-existing valve dysfunction or HF. Changes in valve gradient that exceed the usual increase due to changing cardiac output should be investigated. Symptoms such as new HF, embolic event, or syncope need urgent assessment. Changes in valve clicks and new murmurs should also trigger investigation. Transthoracic and transoesophageal echocardiography, fluoroscopy, and CT can all be used to assess MHV leaflet movement.

Valve thrombosis has a high maternal mortality. Its management is comparable with management in non-pregnant women. In a subacute setting, optimizing anticoagulation with UFH and re-establishing a therapeutic INR with VKA may be sufficient. Thrombolysis may be considered, especially in non-critically ill women, when surgery is not immediately available for critically ill women, and in right-sided prosthetic valve thrombosis.[698] Urgent intervention is often required in acute thrombosis with obstruction or severe regurgitation.[722] This is associated with high maternal and foetal risks. There is a clear survival benefit for the foetus without increasing maternal mortality if cardiac surgery is performed after caesarean section. Therefore, the optimal treatment strategy, as determined by the Pregnancy Heart Team, will depend on type of valve involved, haemodynamic stability of the mother, and gestational age.[421]

12.5.3.2.3. Anticoagulation during delivery in women with mechanical heart valves. The management of delivery for women with MHV is discussed in *Section 4.5.6*. These are high-risk deliveries with significant bleeding complications.

Recommendation Table 19 — Recommendations for prosthetic valves and pregnancy (see Evidence Table 18)

Recommendations	Class[a]	Level[b]
A bioprosthetic valve is recommended (over a mechanical valve) in young women contemplating pregnancy requiring a valve prosthesis.[177]	I	B
It is recommended that the type of valve surgery or intervention for a woman contemplating pregnancy is chosen in consultation with the Pregnancy Heart Team.	I	C
Women with mechanical heart valves		
It is recommended that a care plan documenting the agreed anticoagulant strategy (including the decision to continue VKAs or converting to therapeutic-dose LMWH in the first trimester) is in place for women of childbearing age with an MHV prior to pregnancy or as soon as pregnancy is recognized.[170]	I	C
It is recommended that pregnant women with an MHV are managed by the Pregnancy Heart Team.[170]	I	C
In pregnant women on VKAs, it is recommended to perform INR monitoring weekly or at a minimum every 2 weeks.	I	C
In pregnant women with MHVs on therapeutic-dose LMWH, it is recommended to check peak anti-factor Xa levels and to target levels according to individualized risk.	I	C
During the second and third trimesters until the 36th week, continuing VKAs should be considered in women with prosthetic heart valves at higher risk of thrombosis.	IIa	C
During the second and third trimesters, continuing LMWH with anti-factor Xa level monitoring and dose adjustment may be considered in women at lower risk of thrombosis.	IIb	C
LMWH is not recommended when anti-factor Xa level monitoring is not available.	III	C

INR, international normalized ratio; LMWH, low-molecular-weight heparin; MHV, mechanical heart valve; VKA, vitamin K antagonist.
[a]Class of recommendation.
[b]Level of evidence.

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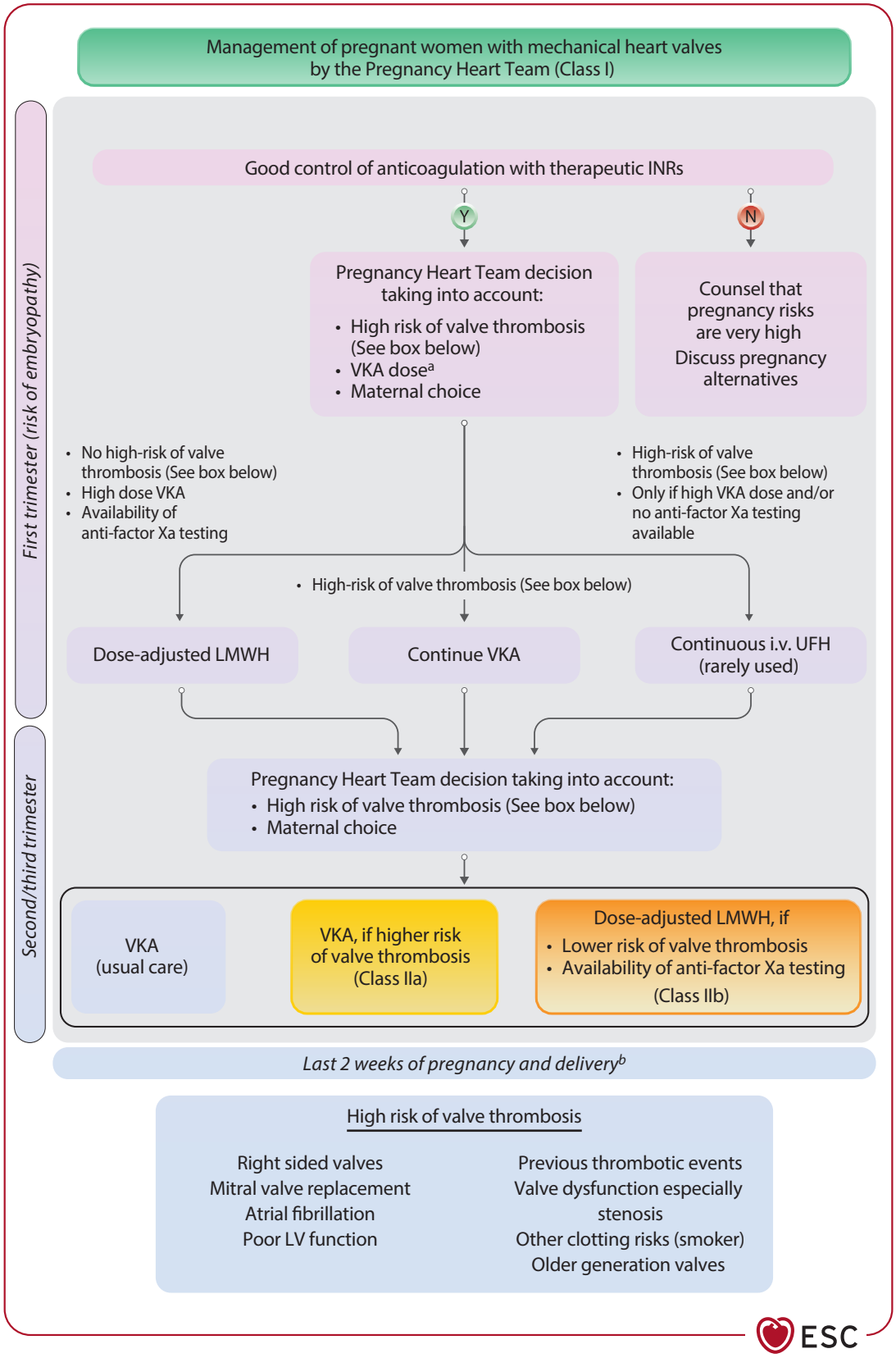
Management of pregnant women with mechanical heart valves by the Pregnancy Heart Team (Class I)
First trimester (risk of embryopathy)
Second/third trimester
Good control of anticoagulation with therapeutic INRs
Y
N
Pregnancy Heart Team decision taking into account:
• High risk of valve thrombosis (See box below)
• VKA dose[a]
• Maternal choice
Counsel that pregnancy risks are very high
Discuss pregnancy alternatives
• No high-risk of valve thrombosis (See box below)
• High dose VKA
• Availability of anti-factor Xa testing
• High-risk of valve thrombosis (See box below)
• Only if high VKA dose and/or no anti-factor Xa testing available
• High-risk of valve thrombosis (See box below)
Dose-adjusted LMWH
Continue VKA
Continuous i.v. UFH (rarely used)
Pregnancy Heart Team decision taking into account:
• High risk of valve thrombosis (See box below)
• Maternal choice
VKA (usual care)
VKA, if higher risk of valve thrombosis (Class IIa)
Dose-adjusted LMWH, if
• Lower risk of valve thrombosis
• Availability of anti-factor Xa testing
(Class IIb)
Last 2 weeks of pregnancy and delivery[b]
High risk of valve thrombosis
Right sided valves
Mitral valve replacement
Atrial fibrillation
Poor LV function
Previous thrombotic events
Valve dysfunction especially stenosis
Other clotting risks (smoker)
Older generation valves
ESC

Figure 21 Management of anticoagulants during the different stages of pregnancy in women with mechanical heart valves. INR, international normalized ratio; i.v., intravenous; LMWH, low-molecular-weight heparin; LV, left ventricle; N, no; UFH, unfractionated heparin; VKA, vitamin K antagonist; Y, yes. [a]See *Table 10*. [b]See *Figure 5* and *Section 4.5.7*.

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12.5.4. Endocarditis prophylaxis

Infective endocarditis during pregnancy is a rare yet severe event associated with maternal and foetal morbidity and mortality. An increasing risk factor is represented by i.v. drug abuse associated with the opioid epidemic in the United States of America.[723] The most common pathogens are *Staphylococcus* (74%) and *Serratia* (13%).[724] A recent comparison across antepartum, delivery, and post-partum maternity-associated infective endocarditis showed the 60-day mortality rate was highest in the delivery subgroup and the rate of valve replacements was highest in post-partum cases.[725] Antibiotics should be given according to guidelines,[178,726] laboratory data on culture and antibiotic sensitivity, and the differential foetal toxicity of antibiotics (see *Section 5*).

12.6. Heart failure

12.6.1. Chronic heart failure

Heart failure complicates 11% of pregnancies in women with pre-existing heart disease and has an in-hospital maternal mortality rate of 9%.[2] Specific pre-pregnancy counselling is needed for women with ventricular dysfunction irrespective of the cause. As many HF medications are contraindicated in pregnancy (see *Section 5*), modifying the drug regimen pre-pregnancy should be part of pregnancy risk stratification, with reassessment after at least 3 months.[727] Contractile reserve off HF therapy, measured by stress echocardiography, can be used for reassessing ventricular function.[371]

Two peaks of HF deterioration occur in pregnancy: at 23–30 weeks and peri-delivery.[182] Pre-conception counselling should include a discussion of management if there is a clinical deterioration during the first peak. Early delivery due to maternal cardiac deterioration will impact foetal outcomes.

Patients with mild ventricular dysfunction may tolerate pregnancy with no increase in symptoms. However, those with worse than mild ventricular dysfunction (mWHO 2.0 class >II) (*Table 6*) require expert care from the Pregnancy Heart Team, with additional input from the advanced HF team including transplant and mechanical circulatory support experts.

Women with pre-existing severe HF (LVEF <30%, mWHO 2.0 class IV) are at high risk of maternal morbidity and mortality and account for up to 15% of maternal deaths globally.[2,728]

Assessment of patients with pre-existing HF includes regular assessment of symptoms, echocardiography, and NP at intervals determined by the severity of HF and other non-cardiac issues.

12.6.2. Acute heart failure (including cardiogenic shock)

Acute heart failure can develop in women without pre-existing heart disease as can be the case in PPCM (*Section 7*), or secondary in women with known cardiac disease such as cardiomyopathy, ischaemic heart disease, ACHD, and severe VHD. The diagnosis can be challenging because the symptoms and signs of AHF can be misinterpreted as changes due to pregnancy (*Figure 22*).[349,729] Pregnant women presenting with AHF require urgent hospital admission. These patients should be referred to an expert centre with established advanced HF care, including on-site surgery and mechanical circulatory support or even a transplant programme as backup.

In case of cardiogenic shock, recommended inotropic agents include levosimendan,[730] dobutamine, and milrinone (*Figure 22*). Levosimendan is administered as a continuous infusion without an initial loading dose. Dobutamine is an option, whereas adrenaline should be avoided. Milrinone may be an alternative if benefits outweigh the risk, due to placental transfer (see Supplementary data online, *Table S2*). Mechanical circulatory support [preferably veno-arterial extracorporeal membrane oxygenation (VA-ECMO)[731]] should be considered in case of severe refractory cardiogenic shock.[339] In cardiogenic shock, urgent delivery by caesarean section with combined spinal/epidural analgesia or general anaesthesia is recommended.[732–734]

Milder cases of acute HF can be treated with oral diuretics, b1-selective beta-blockers (bisoprolol, metoprolol succinate), hydralazine, and oral nitrates. Diuretics (loop diuretics and thiazides if required) should be used with caution due to a potential reduction in uterine blood flow, but may be necessary in pulmonary congestion or echocardiographic signs of high LV end-diastolic pressure.[732]

Recommendation Table 20 — Recommendations for chronic and acute heart failure and pregnancy (see Evidence Table 19)

Chronic heart failure	Class[a]	Level[b]
It is recommended that women with HFrEF are advised about the risk of deterioration of cardiac function during pregnancy and peripartum.[34]	I	C
In pregnant women with HFrEF, it is recommended that non-selective beta-blockers are switched to beta-1-selective blockers (metoprolol, bisoprolol) with close maternal and foetal monitoring.[733,734,738,739]	I	C
Anticoagulation with therapeutic doses of LMWH is recommended in pregnant women with intracardiac thrombus or decreased LV function with EF <35%.[216]	I	C
It is recommended to optimize HF guideline-directed medical therapy after delivery, taking contraindicated drugs during lactation into account[c].[339,734]	I	C
Due to the high metabolic demands of lactation, avoiding lactation may be considered in women with severe HF.[360,733]	IIb	C
ACE-Is, ARBs, ARNIs, MRAs, ivabradine, and SGLT2 inhibitors are not recommended during pregnancy due to adverse foetal effects.	III	C
Acute heart failure		
Inotropes and/or vasopressors are recommended in pregnant women with cardiogenic shock with levosimendan, dobutamine, and milrinone as recommended agents.[730]	I	C
Urgent delivery with caesarean section is recommended in pregnant women with cardiogenic shock as soon as the foetus is viable, taking gestational age, comorbidities, and the available level of medical care into account.[689]	I	C
Early transfer of pregnant women in cardiogenic shock to a facility providing mechanical circulatory support should be considered.[345,740]	IIa	C
Preventing lactation may be considered in women with severe HF due to the high metabolic demands of lactation.[360]	IIb	B

ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor/neprilysin inhibitor; EF, ejection fraction; HF, heart failure; HFrEF, heart failure with reduced ejection fraction; LMWH, low-molecular-weight heparin; LV, left ventricle; MRA, mineralocorticoid receptor antagonist; SGLT2, sodium–glucose co-transporter-2.

[a]Class of recommendation.

[b]Level of evidence.

[c]See *Figure 6* and Supplementary data online, *Table S2*.

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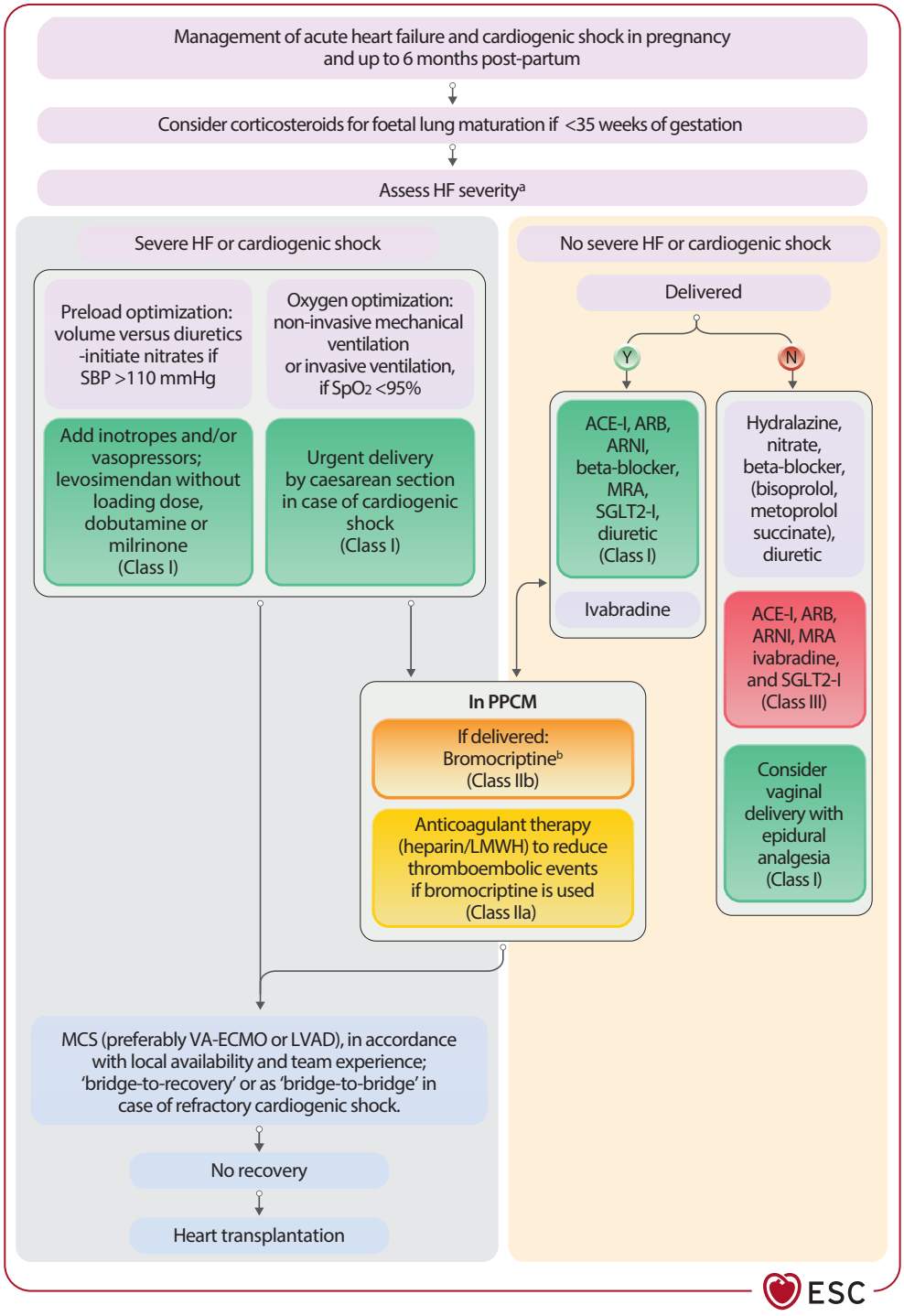

Figure 22 Management of acute heart failure and cardiogenic shock in pregnancy and up to 6 months post-partum. ACE-I, angiotensin-converting enzyme inhibitors; ARB, angiotensin receptor blockers; ARNI, angiotensin receptor/neprilysin inhibitor; b.p.m., beats per minute; HF, heart failure; HR, heart rate; LMWH, low-molecular-weight heparin; LVAD, left ventricular assist device; MCS, mechanical circulatory support; MRA, mineralocorticoid receptor antagonist; PPCM, peripartum cardiomyopathy; SGLT2-I, sodium–glucose co-transporter-2 inhibitor; SBP, systolic blood pressure; SpO_2, oxygen saturation; VA-ECMO, veno-arterial extracorporeal membrane oxygenation. [a]Assessment of heart failure severity: SBP <90 mmHg; HR >130 b.p.m. or <45 b.p.m.; respiratory rate >25/min; SpO_2 <90%; Blood lactate >2.0 mmol/L; low central–venous oxygen saturation <60% (if available); altered mental state; cold, clammy, mottled skin; oliguria <0.5 mL/kg/h. [b]Bromocriptine: starting dose 2.5 mg twice daily with up-titration if needed.

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Beta-blockers should be initiated and gradually up-titrated to the maximum tolerated dose.[345,357,732–734] ACE-Is, ARBs, ARNIs, MRAs, ivabradine, SGLT2 inhibitors, and atenolol are contraindicated during pregnancy due to adverse effects on the foetus. Hydralazine and nitrates appear safe.[43,345,357,733,734]

In the post-partum period, the use of ivabradine to control the heart rate may be considered in addition to beta-blockers, or if there is a contraindication for beta-blockers.[43,339,735,736] In HF with a reversible cause, HF treatment in accordance with guidelines should be recommended for at least 12 months after full LV recovery, followed by gradual tapering[350] (see also *Section 7*). Women with pregnancy-associated HF should be counselled about the risks of recurrence in subsequent pregnancies.[737]

12.7. Special populations

12.7.1. Heart transplantation

Successful pregnancies have been reported in women with heart transplantation, but these pregnancies carry a higher risk. Before transplantation and before a post-transplantation pregnancy, women should be counselled about maternal and foetal risks, including the risks of rejection, infection, graft dysfunction, and potential teratogenicity of drugs. Based on the individual risk factors, it is recommended to postpone pregnancy until at least 1 year after transplantation.[275] Women with heart transplantation are at higher risk of experiencing cardiometabolic complications during pregnancy such as pre-eclampsia, gestational diabetes, hypertension, decreased kidney function, and infections.[741–745] The risk of complications is lower in women with normal graft function and no signs of rejection. Pre-pregnancy evaluation should include standard assessments and, if clinically indicated, coronary angiography, right-heart catheterization, and endomyocardial biopsy.[741–745]

All drugs should be reviewed before pregnancy[275] and mycophenolic acid therapy[275,741] should be avoided due to the teratogenic risk. In cases where the father has the same human leucocyte antigen (HLA) as the donated heart, there is a risk of developing donor-specific antibodies.[746,747] Therefore, consideration should be given to performing paternal HLA testing before conception.

During pregnancy, changes in maternal metabolism can affect the serum drug levels of immunosuppression therapy.[748] Close monitoring of serum drug levels is recommended.[275] Echocardiographic evaluations are recommended to assess graft function during pregnancy. Foetal growth should be regularly evaluated due to the risk of lower birth weight.[745] Vaginal delivery should be considered in stable patients.[745]

Close monitoring of cardiac function, immunosuppression, and donor-specific antibodies is necessary until 6–12 months after delivery due to the risk of post-pregnancy rejection.[275,741,749–751] Lactation may be possible in patients on immunosuppressive drugs, depending on the type of medication (see *Section 5.2.10*).

Recommendation Table 21 — Recommendations for heart transplantation and pregnancy

Recommendations	Class[a]	Level[b]
It is recommended to postpone pregnancy until at least 1 year after heart transplantation, taking individual risk factors into account.[275,741]	I	C
In women with a heart transplant, it is recommended that immunosuppression serum drug levels are monitored during pregnancy every 4 weeks until the 32nd week, then every 2 weeks until the 36th week, then weekly until delivery, and for 6–12 months after delivery to guide dosing.[275]	I	C
It is recommended to perform weekly monitoring of donor-specific antibodies for at least 6–12 months after delivery.[275,741,749–751]	I	C
Paternal HLA testing prior to conception should be considered due to the risk of developing donor-specific antibodies.[746,747]	IIa	C
Mycophenolic acid therapy is not recommended in pregnancy and should be discontinued 6 weeks before conception.[275,741]	III	C

HLA, human leucocyte antigen.
[a]Class of recommendation.
[b]Level of evidence.

12.7.2. Cardio-oncology

12.7.2.1 Gestational cancer

Gestational cancer is defined as cancer that occurs during pregnancy or within 12 months after delivery. The occurrence is ~1 in 1000 pregnancies, with a possible rise in incidence as maternal childbearing age increases. The most frequent cancers affecting pregnant women are breast cancer, cervical and ovarian cancer, lymphoma, leukaemia, colorectal cancer, and melanoma.[752–754] Women diagnosed with cancer during pregnancy should be evaluated and considered by a multidisciplinary team. Both the effect of pregnancy on the cancer and the effect of the cancer on the pregnancy should be evaluated. Patients should be followed in centres with neonatal units in case of premature delivery.

The baseline assessment prior to chemotherapy, and the monthly or bimonthly follow-up during chemotherapy, should include clinical history, physical examination, ECG, cardiac biomarkers, and TTE in the context of physiological haemodynamic changes during pregnancy.[107,755,756] LVEF, NP serial measurements,[756] and high-sensitivity cTn can be used for monitoring of cancer-therapy-related cardiac dysfunction (CTRCD).[734,756]

Chemotherapy given during the first trimester is associated with a high risk of malformation (up to 20%) and miscarriage. Chemotherapy is often avoided after 34 weeks of gestation to provide a ≥3 week window between the last chemotherapy and delivery. Cytotoxic chemotherapies provide different risk profiles during the second and third trimesters.[752,754] The suggested chemotherapy treatments for pregnant women with cancer are summarized in the 2022 ESC Guidelines on cardio-oncology.[757]

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12.7.2.2 Pregnancy in cancer survivors

Improved cancer treatments have led to an increased number of women with pregnancies after oncological therapy. Previous treatment with anthracycline chemotherapy or chest radiotherapy implies a 15-fold increased lifetime risk of developing HF. Furthermore, both the cancer and the cancer treatment have an impact on fertility, pregnancy outcomes, and cardiovascular health. The major risk factors for cardiovascular events during pregnancy in cancer survivors include CTRCD (28%; 47.4 times higher odds), younger age at cancer diagnosis,[758,759] higher cumulative doses of anthracycline, and greater duration between cancer treatment and first pregnancy.[758,759]

Recommendation Table 22 — Recommendations for cardio-oncology and pregnancy

Recommendations	Class[a]	Level[b]
It is recommended that pregnant women with cancer who require cardiotoxic cancer therapy are jointly managed by the Pregnancy Heart Team and the cardio-oncology team.[43]	I	C
Cardiac troponin and NP measurements may be considered at baseline and during anthracycline chemotherapy in pregnant women with cancer.[734,756]	IIb	C

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NP, natriuretic peptide.
[a]Class of recommendation.
[b]Level of evidence.

13. Adverse pregnancy outcomes and long-term management

Adverse pregnancy outcomes (APO), including gestational hypertensive disorders, pre-eclampsia, gestational diabetes mellitus (GDM), small or large for gestational age babies, or pre-term birth are a group of interrelated disorders that share common pathways.[760] Placental dysfunction and oxidative stress in the context of cardiometabolic, genetic, or environmental risk factors may cause APOs.[761] A multidisciplinary care approach is essential (*Figure 23*)[761,762] for optimizing modifiable cardiovascular risk factors after all APOs.[761,762]

13.1. Adverse pregnancy outcomes

13.1.1. Post-partum hypertensive disorders

Hypertension in the post-partum period is most common in women with antenatal hypertensive disorders, but it can also develop *de novo* in the post-partum period. New-onset or *de novo* post-partum pre-eclampsia is increasingly recognized as an important contributor to maternal morbidity and mortality in the post-partum period (*Figure 24*).[763]

Women with pregnancy-related hypertensive disorders, most notably pre-eclampsia, have a higher incidence of several CVDs, including CAD, stroke, and HF.[762,764] Treatment for uncomplicated post-partum hypertension (first 6 weeks after delivery) includes nifedipine and labetalol (metoprolol if labetalol is unavailable).[764] A small randomized controlled trial showed that administration of furosemide in the first 5 days post-partum in women with gestational hypertension and pre-eclampsia significantly reduced the prevalence of persistently elevated BP at 7 days.[765] Methyldopa should be avoided because of the risk of post-partum depression.[602,766] Agents used for the management of acute, severe hypertension in the post-partum period are similar to those used during pregnancy and include labetalol, hydralazine, and nifedipine (see *Sections 5* and *12*).[767] For women with persistent hypertension, antihypertensive therapy should be initiated with reference to lactating status following current guidelines (see *Section 5*).[584,585,767]

13.1.2. Gestational diabetes mellitus

Gestational diabetes mellitus is characterized by glucose intolerance that is first recognized during pregnancy.[768] Haemoglobin A1c testing may help to identify women at high risk.[768,769] Women with GDM have a sharply increased risk of developing type 2 diabetes later in life and a significantly higher likelihood of experiencing adverse cardiovascular events.[770–772] Women with GDM are recommended to undergo a formal oral glucose tolerance test (oGTT) 6–12 weeks post-partum with a repeat assessment at 6–12 months (*Figure 25*). Thereafter, regular annual follow-up with glucose tolerance monitoring is recommended.[773–775]

13.1.3. Pre-term birth

Pre-term delivery occurs after 20 and before the completion of 37 weeks of gestation, regardless of birth weight. There are strong associations between pre-term delivery (alone or with pregnancy-related hypertensive disorders), CVD, and mortality.[771,772] Women with pre-term deliveries are more likely to have an increasing BP trajectory after pregnancy and to be affected by accelerated atherosclerosis independent of traditional CVD risk factors.[776,777] The earlier pre-term delivery occurs, the more strongly it is associated with hypertension and CVD in later life. The relationship of pre-term birth to type 2 diabetes mellitus and dyslipidaemia is inconsistent.

13.1.4. Small for gestational age

Delivering a SGA baby (weight <10th percentile) has not been consistently related to an increased CVD risk, but has been associated with hypertension and diabetes.[761,771,772] Conversely, large for gestational age babies (weight >90th percentile) have been reported to increase maternal CVD risk, but the evidence is limited.[761]

13.1.5. Pregnancy loss and placental abruption

Placental abruption, spontaneous pregnancy loss or stillbirth, and foetal loss after 28 weeks of gestation have been associated with an increased risk of future CVD, with a particularly high risk of hypertension and type 2 diabetes mellitus.[771,778] One or more terminations are associated with a higher CVD risk, however, with limited evidence.[779]

13.2. Breastfeeding

Breastfeeding fosters the recovery of maternal physiological systems to their pre-conception state. Breastfeeding women have a better cardiometabolic profile, and breastfeeding up to 12 months after childbirth has been shown to lower future risk of CVD and mortality,[780–783] potentially due to lowered BP.[784–787] Longer breastfeeding periods are associated with better CVD outcomes.[782,784,788–790] The role of breastfeeding for women who experienced an APO is less clear, but it also appears to have beneficial effects.[789,791,792] There is inconclusive evidence about the beneficial effects of breastfeeding on the cardiovascular health of older women (i.e. aged ≥55 years).[720,788,793,794]

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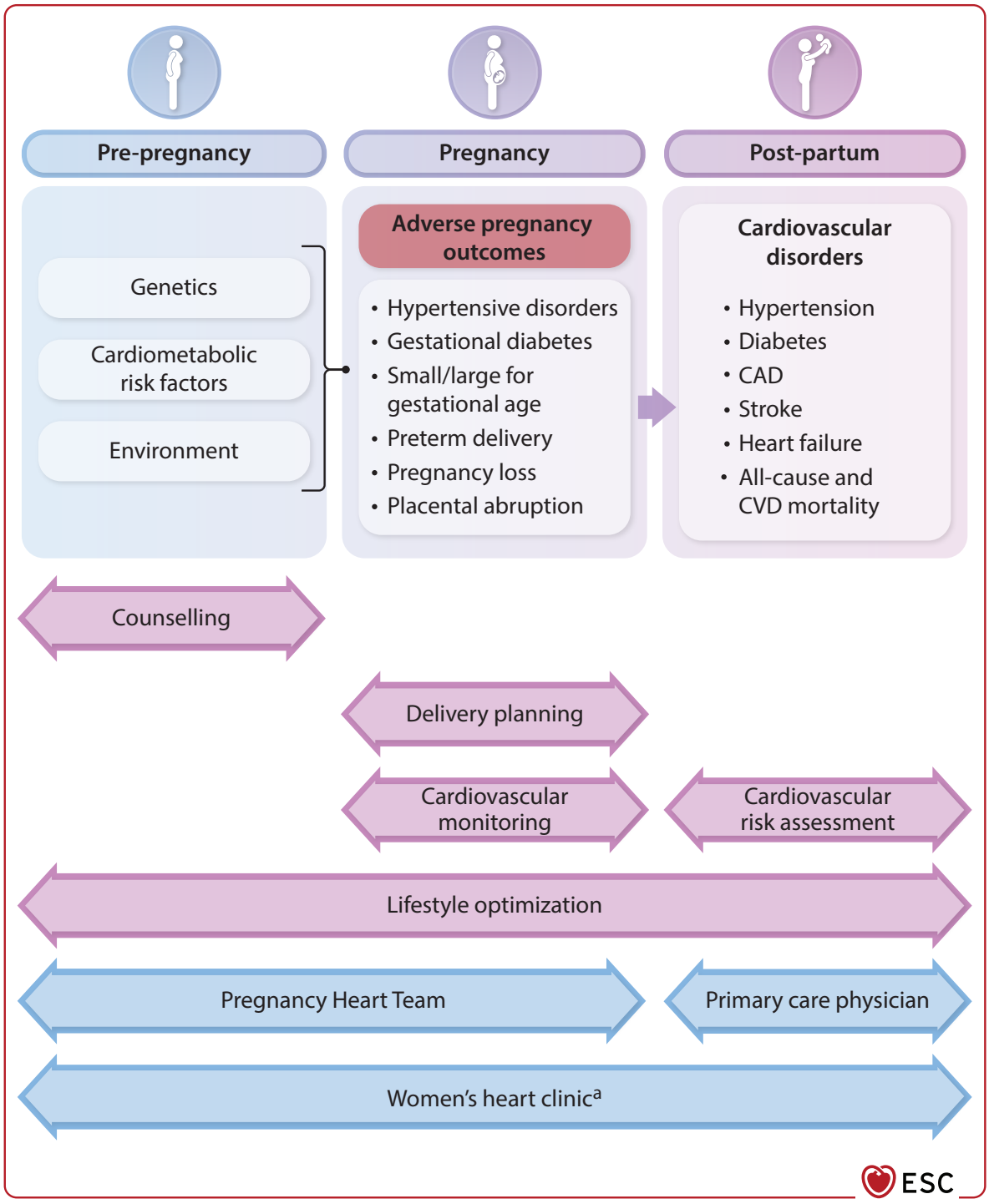

Figure 23 Multidisciplinary approach of adverse pregnancy outcomes. CAD, coronary artery disease; CVD, cardiovascular disease. [a]See *Section 13.3* on Women's Heart Clinics.

13.3. Women's Heart Clinics

Post-partum care is often segmented and only carried out by obstetricians. A longer duration of post-partum care, including cardiovascular risk assessment and counselling on CVD risk prevention, is likely to lower the long-term risk of CVD in women with an APO (*Figure 25*).[762,795] Pregnancy Heart Teams and the potential establishment of Women's Heart Clinics focusing on women of all ages with CVD are necessary to span the care up to post-partum.[795] Seamless communication between the various healthcare providers (e.g. obstetrician, cardiologist, internist, family physician) and multidisciplinary management of APOs is critical for long-term care and the woman's future health.[761,796,797]

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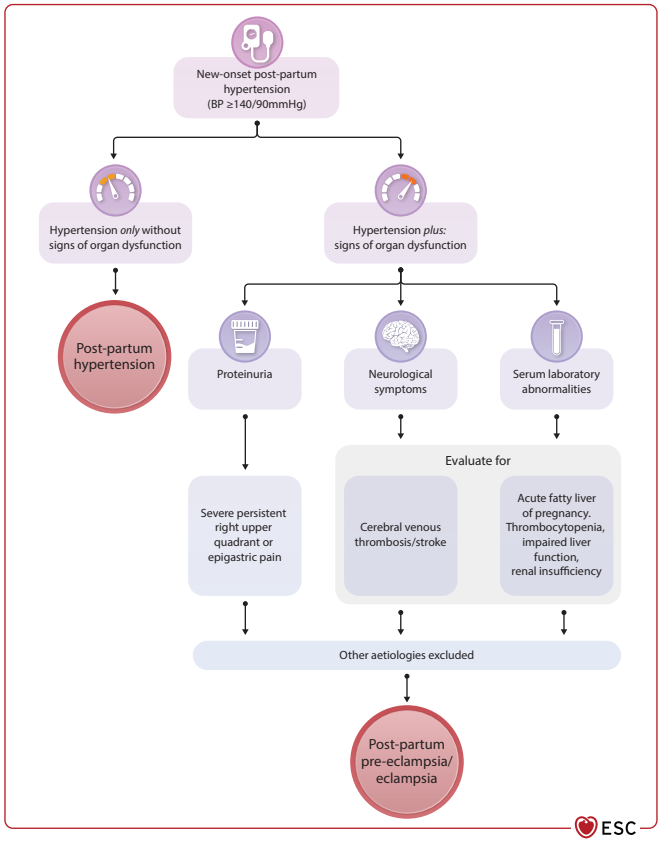
New-onset post-partum hypertension (BP ≥140/90mmHg)
Hypertension *only* without signs of organ dysfunction
Hypertension *plus*: signs of organ dysfunction
Post-partum hypertension
Proteinuria
Neurological symptoms
Serum laboratory abnormalities
Evaluate for
Severe persistent right upper quadrant or epigastric pain
Cerebral venous thrombosis/stroke
Acute fatty liver of pregnancy. Thrombocytopenia, impaired liver function, renal insufficiency
Other aetiologies excluded
Post-partum pre-eclampsia/ eclampsia
ESC

Figure 24 Algorithm for the management of new-onset post-partum hypertension. BP, blood pressure.

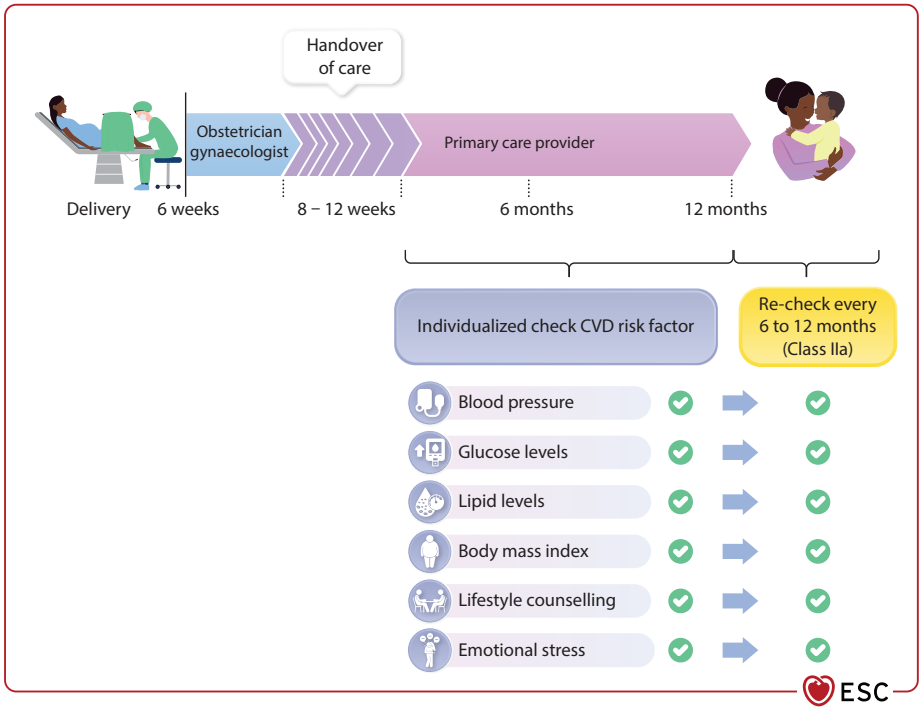

Figure 25 Algorithm for the management of adverse pregnancy outcomes. CVD, cardiovascular disease.

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Recommendation Table 23 — Recommendations for long-term effects of adverse pregnancy outcomes (see Evidence Tables 20 and 21)

Recommendations	Class[a]	Level[b]
It is recommended to undertake a cardiovascular risk assessment in women with APOs, to recognize and document APOs when CVD risk is evaluated in women, and to provide counselling on the importance of healthy lifestyle choices that optimize cardiovascular health.[771,772]	I	B
In women with persistent post-partum hypertension beyond 6 weeks to 3 months post-partum, initiation of antihypertensive therapy with reference to lactating status is recommended following current guidelines.[584,602,798]	I	B
In cases where adoption of healthy lifestyle choices alone is inadequate to control post-partum glucose levels, initiation of pharmacotherapy following current guidelines is recommended.[773,774,799]	I	C
It is recommended that women with a history of GDM undergo a formal oGTT 6–12 weeks post-partum with a repeat assessment at 6–12 months and regular annual follow-up visits to screen for diabetes.[773,774,800]	I	C
Nifedipine and labetalol (metoprolol if labetalol is unavailable) are recommended as treatments for uncomplicated post-partum hypertension in the first 6 weeks after delivery.[602,766,767]	I	C
In women with a history of any APO, cardiovascular risk assessment should be considered at 3 months post-partum with repeat assessment at 6–12 months after implementation of appropriate lifestyle interventions, and regular long-term follow-up thereafter.[761,795]	IIa	C
Breastfeeding may be considered in order to lower the future cardiovascular risk in women with APOs.[789,791,792]	IIb	C

APO, adverse pregnancy outcomes; CVD, cardiovascular disease; GDM, gestational diabetes mellitus; oGTT, oral glucose tolerance test.
[a]Class of recommendation.
[b]Level of evidence.

14. Key messages

- A Pregnancy Heart Team should be involved in the risk assessment, counselling and management of women in mWHO 2.0 class ≥II–III from pre-conception to the late post-partum. Each woman should have a detailed delivery plan agreed in advance.
- In women with known CVD, a complete clinical re-evaluation should take place pre-pregnancy to estimate risk, optimize treatment, consider and evaluate the removal of contraindicated drugs, and reduce the probability of complications.
- Women and their partner (if any) should be offered structured psychosocial support during the entire trajectory, especially for those at high risk and those considering pregnancy termination.
- Women with known heritable cardiovascular disorders should be counselled about the transmission risk, including the option for assisted reproductive technology.
- Management of women with CVD who are pregnant or wishing to become pregnant should be individualized and performed according to a shared decision-making model, respecting the woman's autonomy.
- Women in mWHO 2.0 class IV should be comprehensively counselled about the very high pregnancy risk, being careful to promote a detailed and transparent dialogue about the heightened maternal and foetal risks associated with pregnancy. A shared decision-making process is essential, allowing for informed choices, including the consideration of pregnancy termination if necessary.
- Vaginal delivery is the first choice for the majority of women with CVD.
- In a life-threatening situation, treatments such as defibrillation, interventions, acute coronary revascularization, mechanical circulatory support, and medication should be the same as in non-pregnant women, irrespective of contraindications.
- The use of non-invasive imaging tests with ionizing radiation during pregnancy should only be performed when the benefits clearly outweigh the maternal and foetal risk, and if the result will significantly modify the medical management.
- In women with LQTS and CPVT, the continuation of beta-blockers throughout pregnancy with monitoring of foetal growth is recommended (atenolol is the only contraindicated beta-blocker). Beta-blockers of choice are propranolol and nadolol.
- In women with LQT2, post-partum is a distinct high-risk period, and therefore full dosage of beta-blockers is strongly recommended.
- Genetic testing should be considered in PPCM.
- In women with PPCM and DCM, subsequent pregnancy is not recommended if LV function does not normalize.
- Genetic testing in women with aortic disease wishing to conceive is recommended and management should be based on the presence and type of P/LP variant.
- Women with the following ACHD lesions should be provided with expert counselling and education by a Pregnancy Heart Team, with clear and thorough discussion of the very high pregnancy risk and the need for a shared decision-making process:
  - Systemic RV, in NYHA class III–IV, ventricular dysfunction (EF <40%), more than moderate TR, or treated HF;
  - A Fontan circulation and oxygen saturation <85%, reduced ventricular function, severe arrhythmias, or in NYHA class III–IV.
- There is no safe cut-off value for elevated pulmonary artery pressure in pregnancy.

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- Women of childbearing potential with PAH should be counselled at the time of diagnosis about the risks and uncertainties associated with becoming pregnant.
- Any suspicion of VTE, including DVT and PE, requires an immediate formal assessment with validated diagnostic tests by a multidisciplinary specialized team.
- LMWH is the agent of choice for prophylaxis and treatment of VTE in pregnancy.
- When treating women with HF during pregnancy, it should be noted that several drugs [ACE-Is, ARBs, direct renin inhibitors, sacubitril–valsartan (ARNIs), MRAs, and SGLT2 inhibitors] are not recommended. When inotropes or more advanced treatment is necessary, referral to an expert centre is recommended.
- When possible, mechanical valves should be avoided in girls and women of childbearing age.
- Methyldopa, labetalol, and CCBs are recommended for the treatment of hypertension in pregnancy.
- Women at high or moderate risk of pre-eclampsia should be advised to additionally take 75–100 mg of ASA daily from weeks 12 to 36/37.
- After cardiac transplantation, it is recommended to postpone pregnancy for at least 1 year, taking individual risk factors into account.
- Women with APOs should be informed about long-term risks and preventive strategies and offered appropriate follow-up, including psychosocial support (if necessary).

15. Gaps in evidence

Pre-pregnancy counselling and evaluation

- Data on the adverse effects of assisted reproductive treatment in women with CVD are lacking.

Diagnostic methods

- There is a lack of data on the safety of echocardiographic contrast agents during pregnancy or lactation.
- There are controversial data on the use of gadolinium-based contrast agents in pregnancy.
- There are no clear cut-offs for NT-proBNP levels during pregnancy.
- There are no normative values of cTnI and cTnT in pregnancy and the post-partum period.
- There is a lack of data on normal lung ultrasound pattern during pregnancy.

Drugs during pregnancy and lactation

- Safety data of DOACs and antidotes (idarucizumab, andexanet alfa, cirapantag) in pregnancy are lacking.
- Safety data of newer anti-arrhythmic drugs and rate-controlling drugs (vernakalant, ivabradine, landiolol) in pregnancy are lacking.

Cardiomyopathy and primary arrhythmia syndromes

- The available data on gene-specific management during pregnancy in different cardiomyopathies and primary arrhythmia syndromes are limited.

Peripartum cardiomyopathy

- The potential for recovery of cardiac function in PPCM remains unclear and the risks in subsequent pregnancies are not well defined.

Aortopathies

- More data are needed to correctly estimate the pregnancy risk in women with previous aortic dissection and/or aortic root surgery.
- Risk factors for aortic dissection in the post-partum period are poorly understood, making counselling about this difficult.
- It is unclear whether a distinction between root and ascending phenotype in women with BAV should lead to a different threshold for prophylactic surgery (as in non-pregnant women).

Congenital heart disease

- More data are needed to estimate the risk and the long-term effects of pregnancy (including multiple pregnancies), especially in women with a Fontan circulation or univentricular hearts.
- Risk factors for the development of heart failure and arrhythmias in pregnant women with (systemic) right-heart failure are poorly understood.

Pulmonary hypertension

- Defining the optimal timing to start or escalate PAH therapies in pregnancy complicated with PAH remains challenging.

Venous thromboembolism

- Data on risk stratification of VTE in pregnancy are limited, specifically in those with other pre-existing comorbidities.
- Data on the use of anticoagulant agents (other than LMWH) are limited, just as data on the efficacy and safety of inferior vena cava filters and catheter-based thrombectomy (in PE).

Acquired heart disease

- The foetal risks associated with the newer HF medications remain unclear, particularly regarding exposure during different trimesters.
- The optimal tools to stratify risk of recurrence for atherosclerotic and SCAD ACS are unknown.
- Physiopathological mechanisms of SCAD in pregnancy are unknown.
- Optimal treatment of SCAD during pregnancy is not well established.
- There is scarce evidence about the necessity of using statins during pregnancy in women with cardiovascular risk or established ASCVD.
- Optimal anticoagulation strategies for women with MHVs during pregnancy remain uncertain.
- The role of anti-factor Xa level monitoring needs to be determined.

Women's Heart Clinics

- Optimal strategies for surveillance and follow-up of women with APOs are unclear.
- It is unclear how social determinants of health (the environmental factors that affect how people live, learn, and work) affect APOs.
- There is a need for studies exploring models of post-natal care, starting from the initial antenatal visit through to the end of the post-partum period.

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- Further research is needed to identify risk factors for pregnancy-related depression and poor health behaviour engagement in women with CVD, enabling the development of tailored interventions to improve their health and quality of life.

16. 'What to do' and 'what not to do' messages from the Guidelines

Table 16 lists all Class I and Class III recommendations from the text alongside their level of evidence.

17. Evidence tables

Evidence tables are available at *European Heart Journal* online.

18. Data availability statement

No new data were generated or analysed in support of this research.

Table 16 'What to do' and 'what not to do'

Recommendations	Class[a]	Level[b]
4. The Pregnancy Heart Team		
Counselling, pregnancy risk assessment, contraception, assisted reproductive technology, and the involvement of a Pregnancy Heart Team		
It is recommended to perform a risk assessment in all women with CVD of childbearing age using the mWHO 2.0 classification.	I	C
A discussion by the Pregnancy Heart Team about the high risk of maternal mortality or morbidity and the related high foetal risk is recommended for women with mWHO 2.0 class IV conditions, including a shared decision-making process for pregnancy termination, involving psychological support.	I	C
It is recommended that women with CVD of mWHO 2.0 class II–III and above are evaluated and managed by a Pregnancy Heart Team from pre-pregnancy onwards through pregnancy and post-partum.	I	C
It is recommended that women with CVD of mWHO 2.0 class II and above, or those at risk of developing CVD, receive individualized advice to determine the most suitable contraception method, including emergency contraception.	I	C
Assessment by a clinical geneticist prior to pregnancy is recommended in women fulfilling diagnostic criteria for inherited cardiovascular disease to guide risk stratification and pre-natal genetic testing.	I	C
Pre-conception genetic counselling is recommended in couples with heritable CVD, whether genetic testing is being considered or not. It is recommended that this counselling is provided by an appropriately trained healthcare professional within a multidisciplinary team that offers psychological support and education to encourage decision-making.	I	C
It is recommended that single embryo transfer is performed in women with CVD.	I	C
It is recommended to offer women with CVD access to termination of pregnancy that is tailored to their cardiac condition to minimize the risks of the procedure.	I	C
Diagnostic methods in pregnancy		
Transthoracic echocardiography is recommended as first-line imaging tool in any pregnant woman with unexplained or new cardiovascular signs or symptoms.	I	C
It is recommended to limit exposure to all medical ionizing radiation doses to ALARA levels.	I	C
It is recommended to keep the radiation dose to the foetus as low as possible (preferably <50 mGy), particularly if the foetus is in the field of view.	I	C
Timing and mode of delivery		
Vaginal delivery is recommended in most women with CVD.	I	B
Routine induction of labour prior to 39 weeks is not recommended in women with stable CVD.	III	C
It is recommended that the timing of delivery is planned to ensure safe and effective peripartum anticoagulation.	I	C
It is recommended to discontinue VKAs and start therapeutic-dose LMWH or adjusted-dose i.v. UFH at the 36th week of gestation or 2 weeks before the planned delivery.	I	C
In women at low risk on therapeutic-dose LMWH, neuraxial anaesthesia and vaginal delivery (or caesarean section for obstetric indications) is recommended 24 h after the last dose of LMWH.	I	C
In women at high risk, it is recommended to convert LMWH to i.v. UFH at least 36 h prior to delivery and stop the UFH infusion 4–6 h prior to anticipated delivery. The aPTT should be normal before regional anaesthesia.	I	C
If delivery starts while the mother is on VKAs or <2 weeks after discontinuation of VKAs, caesarean section is recommended for foetal protection.	I	C
Post-delivery, it is recommended that the decision to restart LMWH or UFH is made after discussion with the Pregnancy Heart Team and the woman who gave birth.	I	C

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It is recommended to postpone the switch from heparin back to oral anticoagulants until 7–14 days post-partum when the wound area has healed, in consultation with the Pregnancy Heart Team.	I	C
5. Drugs during pregnancy and lactation		
Direct oral anticoagulants and pregnancy		
DOACs are not recommended during pregnancy.	III	C
6. Pregnancy in women with cardiomyopathies and primary arrhythmia syndromes		
Cardiomyopathies and pregnancy		
Clinical cardiological surveillance (ECG, echocardiogram, and Holter ECG monitoring) is recommended during pregnancy in women with CMPs, depending on individual risk.	I	C
Vaginal delivery is recommended in most women with CMPs, unless there are obstetric indications for caesarean section, severe HF (EF <30% and/or NYHA class III/IV), uncontrolled arrhythmias, or severe outflow obstruction (≥50 mmHg) in women with HCM, or in women presenting in labour on VKAs.	I	C
In women with DCM and worsening of EF during pregnancy, counselling on the risk of recurrence during a subsequent pregnancy is recommended in all cases, even after recovery of LV function.	I	C
It is recommended to use the same risk stratification protocol for ventricular arrhythmias in pregnant women with HCM as for non-pregnant women with HCM.	I	C
It is recommended to start beta-blockers in women with HCM who develop symptoms due to outflow tract obstruction or arrhythmia during pregnancy.	I	C
It is recommended that women with HCM with symptomatic LV dysfunction (EF <50%) and or severe LVOTO (≥50 mmHg) wishing to become pregnant are counselled by the Pregnancy Heart Team regarding the high risk of pregnancy-related adverse events.	I	C
Myosin inhibitors are not recommended in women during pregnancy due to lack of safety data.	III	C
Primary arrhythmia syndromes and pregnancy		
Monitoring and treatment of hypokalaemia and hypomagnesaemia is recommended in pregnant women with primary arrhythmia syndromes suffering from hyperemesis.	I	C
Beta-blockers, with pre-pregnancy dose and with nadolol and propranolol as drugs of choice, are recommended during pregnancy in women with LQTS.	I	B
It is recommended to continue beta-blocker therapy during lactation in women with LQTS to reduce arrhythmic risk.	I	B
Pre-pregnancy dose beta-blocker of nadolol or propranolol are recommended in women with LQT2, particularly in the post-partum period, which represents a high-risk period for life-threatening arrhythmias.	I	B
Beta-blockers, with pre-pregnancy dose and with nadolol and propranolol as drugs of choice, are recommended during pregnancy and lactation in women with CPVT.	I	C
Flecainide, in addition to beta-blockers, is recommended in women with CPVT who experience cardiac events such as syncope, VT, or cardiac arrest, during pregnancy.	I	C
It is recommended that women with CPVT who are stable on beta-blockers (nadolol or propranolol as drugs of choice) and flecainide before pregnancy, continue both drugs also during pregnancy and post-partum.	I	C
7. Peripartum cardiomyopathy		
Counselling for women with PPCM about the risk of recurrence during a subsequent pregnancy and about contraception is recommended in all cases, even after recovery of LV function (LVEF >50%).	I	C
8. Pregnancy in women with aortopathies		
It is recommended that women with aortic disease have counselling about the risk of aortic dissection in pregnancy and the post-partum period.	I	C
It is recommended that women with a history of aortic dissection or -surgery have pre-pregnancy counselling about the high risk by an extended Pregnancy Heart Team considering the presence and type of genetic variant, aortic morphology, growth rate, and aetiology of aortic dissection.	I	C
It is recommended that women with vascular Ehlers–Danlos syndrome wishing to become pregnant are counselled regarding the very high risk of pregnancy-related adverse events by a multidisciplinary team, considering family history, genetic variant, and previous vascular events.	I	C
Imaging of the entire aorta (CT or CMR) is recommended before pregnancy in women with known or suspected aortic disease.	I	C
In women with aortic dilatation related to BAV, imaging (with TTE, and CMR/CT if needed) of the aortic root, ascending aorta, and descending aorta (to rule out coarctation) is recommended before pregnancy.	I	C
In women with low-risk aortic disease (mWHO 2.0 classes II and II–III), one-time echocardiographic imaging between 20 and 30 weeks of gestation and imaging at 6 months post-partum is recommended.	I	C

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In women with moderate to high-risk aortic disease (mWHO 2.0 classes III and IV), repeated echocardiographic imaging every 4–12 weeks (depending on diagnosis and severity of dilatation) is recommended during pregnancy and until 6 months post-partum.	I	C
CMR (without gadolinium) imaging of the entire aorta is recommended in pregnant women at risk of or with known aortic dilatation who have not had recent pre-pregnancy cross-sectional imaging.	I	C
It is recommended that centres managing pregnancies in women with moderate to high-risk aortic disease (mWHO 2.0 class III/IV) can provide cardiovascular surgery in case of peripartum adverse events.	I	C
When a woman with known aortic dilatation, history of dissection, or P/LP variant associated with aortic disease becomes pregnant, strict and individualized BP control is recommended.	I	C
Beta-blocker therapy throughout pregnancy and in the post-partum period is recommended in women with MFS and other HTADs.	I	C
Celiprolol is recommended in women with vascular Ehlers–Danlos syndrome during pregnancy and lactation.	I	C
It is recommended that indications for pre-pregnancy aortic root and/or ascending aortic surgery are guided by aortic morphology, underlying pathology, family history, genetic variant, previous vascular events, and patient's preference.	I	C
In women with MFS and aortic root diameters >45 mm, surgery before pregnancy is recommended.	I	C
In women with LDS with P/LP variants in *TGFBR1*, *TGFBR2*, and aortic root diameters ≥45 mm, surgery before pregnancy is recommended.	I	C
In women with nsHTAD with P/LP variants in *MYH11*, *ACTA2*, *PRKG1*, or *MYLK*, and aortic root diameters ≥45 mm, surgery before pregnancy is recommended.	I	C
In women with BAV and aortic root or ascending aortic diameter ≥50 mm, surgery before pregnancy is recommended.	I	C
In women without an identifiable P/LP variant with aortic root or ascending aortic diameters ≥50 mm, surgery before pregnancy is recommended.	I	C
In women with an aorta <40 mm, vaginal delivery is recommended.	I	C
In women with vascular Ehlers–Danlos syndrome, caesarean section at 37 weeks is recommended for obstetrical reasons.	I	C
The use of ergometrine post-delivery is not recommended in women with aortopathy.	III	C
9. Pregnancy in women with known congenital heart disease		
Vaginal delivery is recommended in most women with ACHD.	I	B
It is recommended that all women with Fontan circulation who wish to become pregnant receive counselling from the Pregnancy Heart Team regarding the high risk of pregnancy-related adverse events.	I	C
It is recommended that women with a systemic RV (Mustard/Senning or congenitally corrected TGA), in NYHA class III/IV, systemic ventricular dysfunction (EF <40%), or severe TR wishing to become pregnant are counselled by the Pregnancy Heart Team regarding the high risk of pregnancy-related adverse events.	I	C
In women with significant haemodynamic lesions, discussion about guideline-directed interventions is recommended prior to pregnancy.	I	C
10. Pregnancy in women with pulmonary arterial hypertension		
It is recommended that women of childbearing potential with PAH wishing to become pregnant are counselled by a multidisciplinary team regarding the very high risk of pregnancy-related adverse events, encouraging a shared decision-making process about whether to become pregnant.	I	C
It is recommended to provide clear contraceptive advice to women of childbearing potential with PAH.	I	C
For women with PAH requiring pregnancy termination, it is recommended to perform this in PH centres.	I	C
Endothelin receptor antagonists, riociguat, and selexipag are not recommended during pregnancy.	III	C
11. Venous thromboembolism in pregnancy and post-partum		
For pregnant or post-partum women at high risk of VTE, a prophylactic fixed dose of LMWH is recommended over a higher weight-adjusted dose to reduce the risk of VTE.	I	B
In pregnant women or women in the post-partum period with suspicion of VTE (DVT and/or PE), an immediate formal diagnostic assessment with validated methods is recommended and should not be postponed.	I	B
In pregnant women or women in the post-partum period with newly diagnosed VTE (DVT and/or PE), the involvement of the Pregnancy Heart Team, including a vascular specialist and a haematologist, is recommended.	I	C
In pregnant or post-partum women with a diagnosis of VTE without haemodynamic instability, anticoagulation is recommended by using therapeutic-dose LMWH based on early pregnancy body weight.	I	C
12. Pregnancy in women with acquired heart disease		
Coronary artery disease and pregnancy		
In pregnant women with chest pain, it is recommended to exclude life-threatening cardiovascular conditions, including PE, ACS (including SCAD), and acute aortic syndrome.	I	C

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It is recommended to manage pregnant women with ACS in the same way as non-pregnant women, including diagnostic investigations and interventions.	I	C
Low-dose ASA is recommended as the antiplatelet treatment of choice during pregnancy and lactation when single antiplatelet treatment is indicated.	I	B
If DAPT is required, clopidogrel is recommended as the P2Y12 inhibitor of choice during pregnancy.	I	C
The duration of DAPT (aspirin and clopidogrel) in pregnant women undergoing coronary stent implantation is recommended to be the same as in non-pregnant women, with an individual approach considering ischaemic risk and delivery-related bleeding risks.	I	C
Hypertensive disorders and pregnancy		
It is recommended to aim for systolic BP <140 mmHg and diastolic BP <90 mmHg in pregnant women.	I	B
Systolic BP ≥160 mmHg or diastolic BP ≥110 mmHg in a pregnant woman is an emergency, and treatment in a hospital setting is recommended.	I	C
Low-dose aspirin (75–150 mg daily) is recommended in women at moderate or high risk of pre-eclampsia (i.e. at least one high risk factor or two moderate risk factors for pre-eclampsia) from weeks 12 to 36/37.	I	A
In women with gestational hypertension, initiation of drug treatment is recommended at systolic BP ≥140 mmHg or diastolic BP ≥90 mmHg.	I	B
Methyldopa is recommended for the treatment of hypertension in pregnancy.	I	B
Labetalol, metoprolol, and dihydropyridine calcium channel blockers are recommended for the treatment of hypertension in pregnancy.	I	C
In severe hypertension, drug treatment with i.v. labetalol, urapidil, nicardipine, or oral short-acting nifedipine or methyldopa is recommended for acute reduction in blood pressure. Intravenous hydralazine is a second-line option.	I	C
In pre-eclampsia associated with pulmonary oedema, nitroglycerine given as an i.v. infusion is recommended.	I	C
In women with pre-eclampsia without severe features, delivery is recommended at 37 weeks.	I	B
It is recommended to expedite delivery in women with pre-eclampsia associated with adverse markers such as haemostatic disorders.	I	C
In women with gestational hypertension, delivery is recommended at 39 weeks.	I	B
Supraventricular tachycardia and pregnancy		
Immediate electrical cardioversion is recommended for acute treatment of SVT with haemodynamic instability.	I	C
Vagal manoeuvres and i.v. adenosine are recommended for conversion of haemodynamically stable supraventricular tachycardias.	I	C
Intravenous beta-blockers (metoprolol) are recommended as the first-line option for acute rate control in pregnant women with AF or AF with preserved LVEF and rapid ventricular rate.	I	C
Therapeutic anticoagulation with LMWH is recommended for pregnant women with persistent or permanent AF at elevated thromboembolic risk.	I	C
Beta-1-selective blockers are recommended for rate control in pregnant women with AF, AFL, or FAT.	I	C
Beta-1-selective blockers or verapamil are recommended for the prevention of SVT in women without pre-excitation on resting ECG.	I	C
Flecainide or propafenone are recommended for the prevention of arrhythmias in pregnant women with WPW syndrome.	I	C
Ventricular tachycardia, device implantation, catheter ablation, and pregnancy		
Immediate electrical cardioversion is recommended for both unstable and stable ventricular tachycardias.	I	C
Beta-blockers or verapamil are recommended for the prevention of idiopathic sustained VT.	I	C
If an ICD, pacemaker, or resynchronization therapy device is indicated during pregnancy, implantation is recommended with optimal radiation protection.	I	C
Cardiac arrest and pregnancy		
Continuous manual left uterine displacement during CPR in pregnant women (≥20 weeks) with cardiac arrest is recommended to relieve aortocaval compression.	I	C
It is recommended to establish i.v. access above the diaphragm to ensure that the i.v. therapy is not obstructed by the gravid uterus.	I	C
It is recommended to perform the same chest compressions and defibrillation protocols in pregnant as in non-pregnant women.	I	C
Anterolateral defibrillator pad placement is recommended with the lateral pad placed under the breast.	I	C
It is recommended that no drugs are withheld in pregnant women with cardiac arrest due to concerns of teratogenicity.	I	C
Congenital atrioventricular block and pregnancy		
In pregnant women with asymptomatic congenital AV block, normal cardiac anatomy and function, a narrow QRS complex, and ventricular rate (≥50 b.p.m.), a prophylactic temporary pacemaker during delivery is not recommended.	III	C
Native valve disease and pregnancy		
Intervention is recommended before pregnancy in symptomatic patients with severe aortic stenosis.	I	C
Intervention is recommended before pregnancy in women with mitral stenosis and a valve area <1.5 cm^2.	I	C

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In pregnant women with symptomatic mitral stenosis or pulmonary hypertension, restricted activities and beta-blockers are recommended.	I	C
In pregnant women with mitral stenosis, diuretics are recommended when congestive symptoms persist despite beta-blockers.	I	C
Full therapeutic-dose anticoagulation is recommended in women with mitral stenosis complicated by AF, left atrial thrombus, or prior embolism.	I	C
Surgical treatment is recommended before pregnancy in women with severe aortic or mitral regurgitation with symptoms, impaired ventricular function, or marked ventricular dilatation.	I	C
Diuretics are recommended in pregnant women with regurgitant lesions when symptoms or signs of congestion occur.	I	C
Prosthetic valves and pregnancy		
A bioprosthetic valve is recommended (over a mechanical valve) in young women contemplating pregnancy requiring a valve prosthesis.	I	B
It is recommended that the type of valve surgery or intervention for a woman contemplating pregnancy is chosen in consultation with the Pregnancy Heart Team.	I	C
It is recommended that a care plan documenting the agreed anticoagulant strategy (including the decision to continue VKAs or converting to therapeutic-dose LMWH in the first trimester) is in place for women of childbearing age with an MHV prior to pregnancy or as soon as pregnancy is recognized.	I	C
It is recommended that pregnant women with an MHV are managed by the Pregnancy Heart Team.	I	C
In pregnant women on VKAs, it is recommended to perform INR monitoring weekly or at a minimum every 2 weeks.	I	C
In pregnant women with MHVs on therapeutic-dose LMWH, it is recommended to check peak anti-factor Xa levels and to target levels according to individualized risk.	I	C
LMWH is not recommended when anti-factor Xa level monitoring is not available.	III	C
Chronic and acute heart failure and pregnancy		
It is recommended that women with HFrEF are advised about the risk of deterioration of cardiac function during pregnancy and peripartum.	I	C
In pregnant women with HFrEF, it is recommended that non-selective beta-blockers are switched to beta-1-selective blockers (metoprolol, bisoprolol) with close maternal and foetal monitoring.	I	C
Anticoagulation with therapeutic doses of LMWH is recommended in pregnant women with intracardiac thrombus or decreased LV function with EF <35%.	I	C
It is recommended to optimize HF guideline-directed medical therapy after delivery, taking contraindicated drugs during lactation into account.	I	C
Urgent delivery with caesarean section is recommended in pregnant women with cardiogenic shock as soon as the foetus is viable, taking gestational age, comorbidities, and the available level of medical care into account.	I	C
Inotropes and/or vasopressors are recommended in pregnant women with cardiogenic shock with levosimendan, dobutamine, and milrinone as recommended agents.	I	C
ACE-Is, ARBs, ARNIs, MRAs, ivabradine, and SGLT2 inhibitors are not recommended during pregnancy due to adverse foetal effects.	III	C
Heart transplantation and pregnancy		
It is recommended to postpone pregnancy until at least 1 year after heart transplantation, taking individual risk factors into account.	I	C
In women with a heart transplant, it is recommended that immunosuppression serum drug levels are monitored during pregnancy every 4 weeks until the 32nd week, then every 2 weeks until the 36th week, then weekly until delivery, and for 6–12 months after delivery to guide dosing.	I	C
It is recommended to perform weekly monitoring of donor-specific antibodies for at least 6–12 months after delivery.	I	C
Mycophenolic acid therapy is not recommended in pregnancy and should be discontinued 6 weeks before conception.	III	C
Cardio-oncology and pregnancy		
It is recommended that pregnant women with cancer who require cardiotoxic cancer therapy are jointly managed by the Pregnancy Heart Team and the cardio-oncology team.	I	C
13. Long-term effects of adverse pregnancy outcomes		
It is recommended to undertake a cardiovascular risk assessment in women with APOs, to recognize and document APOs when CVD risk is evaluated in women, and to provide counselling on the importance of healthy lifestyle choices that optimize cardiovascular health.	I	B
In women with persistent post-partum hypertension beyond 6 weeks to 3 months post-partum, initiation of antihypertensive therapy with reference to lactating status is recommended following current guidelines.	I	B
In cases where adoption of healthy lifestyle choices alone is inadequate to control post-partum glucose levels, initiation of pharmacotherapy following current guidelines is recommended.	I	C
It is recommended that women with a history of GDM undergo a formal oGTT 6–12 weeks post-partum with a repeat assessment at 6–12 months and regular annual follow-up visits to screen for diabetes.	I	C

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Nifedipine and labetalol (metoprolol if labetalol is unavailable) are recommended as treatments for uncomplicated post-partum hypertension in the first 6 weeks after delivery.	I	C

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ACE-I, angiotensin-converting enzyme inhibitor; ACHD, congenital heart disease; ACS, acute coronary syndrome; AF, atrial fibrillation; AFL, atrial flutter; ALARA, as low as reasonably achievable; aPTT, activated partial thromboplastin time; APO, adverse pregnancy outcome; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor/neprilysin inhibitor; ASA, acetylsalicylic acid; AV, atrioventricular; BAV, bicuspid aortic valve; BP, blood pressure; CMP; cardiomyopathy; CMR, cardiovascular magnetic resonance; CPR, cardiopulmonary resuscitation; CPVT, catecholaminergic polymorphic ventricular tachycardia; CT, computed tomography; CVD, cardiovascular disease; DAPT, dual antiplatelet therapy; DCM, dilated cardiomyopathy; DOAC, direct oral anticoagulant; DVT, deep vein thrombosis; ECG, electrocardiogram; EF, ejection fraction; FAT, focal atrial tachycardia; GDM, gestational diabetes mellitus; HCM, hypertrophic cardiomyopathy; HF, heart failure; HFrEF, heart failure with reduced ejection fraction; HTAD, heritable thoracic aortic disease; ICD, implantable cardioverter defibrillator; INR, international normalized ratio; i.v., intravenous; LDS, Loeys–Dietz syndrome; LMWH, low-molecular-weight heparin; LQT2, long QT syndrome type 2; LQTS, long QT syndrome; LV, left ventricle; LVEF, left ventricular ejection fraction; LVOTO, left ventricular outflow tract obstruction; MFS, Marfan syndrome; MHV, mechanical heart valve; MRA, mineralocorticoid receptor antagonist; mWHO, modified World Health Organization; nsHTAD, non-syndromic heritable thoracic aortic disease; NYHA, New York Heart Association; oGTT, oral glucose tolerance test; PAH, pulmonary arterial hypertension; PE, pulmonary embolism; PH, pulmonary hypertension; P/LP, pathogenic/likely pathogenic; PPCM, peripartum cardiomyopathy; RV, right ventricle; SCAD, spontaneous coronary artery dissection; SGLT2, sodium–glucose co-transporter-2; SVT, supraventricular tachycardia; TGA, transposition of the great arteries; TR, tricuspid regurgitation; TTE, transthoracic echocardiogram; UFH, unfractionated heparin; VKA, vitamin K antagonist; VT, ventricular tachycardia; VTE, venous thromboembolism; WPW, Wolff–Parkinson–White.

[a]Class of recommendation.

[b]Level of evidence.

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20. Appendix

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